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OF

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Original Investigations of Cattle Diseases in Nebraska, 1886–1889. By FRANK S. BILLINGS, while Director of the Patho-Biological Laboratory of the State University of Nebraska.

JUNE, 1889.

LINCOLN, NEBRASKA,
U. S. A.

ORIGINAL INVESTIGATIONS

OF

CATTLE DISEASES

IN

NEBRASKA,

1886-1888.

BY

FRANK S. BILLINGS,

WHILE DIRECTOR OF THE PATHO-BIOLOGICAL LABORATORY OF THE STATE UNIVERSITY OF NEBRASKA.

(Bulletins 7, 8, 9, and 10.)

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PREFATORY NOTE.

This volume, consisting of Bulletins 7, 8, 9 and 10 of the Agricultural Experiment Station of Nebraska, is issued in this form in accordance with a resolution of the Board of Control, passed April 10th, 1889, in order that the results of the work of the Investigator of Animal Diseases may be placed permanently before the stock-growers of the State.

CHARLES E. BESSEY,
Director.

June 5, 1889.

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NOMENCLATURE.

The nomenclature of this disease has been as varied as the ignorance of people about it. It has been called "murrain," which means no more than an extended and fatal disease; the "splenic fever," which only expresses the fact that one of its pathological symptoms is an enlarged spleen, a phenomenon by no means peculiar to it, as we find the same in anthrax and other diseases; "Texas fever," because most frequently caused in the North through cattle from that State; "Spanish fever," because it was assumed to have been introduced into the country by the early Spanish settlers; "cattle fever," which means nothing; and "periodic fever," which means still less.

The word "fever," to express any peculiar characteristic of any given disease, is a misnomer, illogical, and unpathological. Its continued use in this respect is one of the most lamentable phenomena in connection with medicine. It shows the most abject ignorance of every principle of pathological philosophy. It is to be absolutely condemned, as it fails to give us any idea of the true nature of any disease. It describes a phenomenon common to almost any irritative disturbance of an animal organism; hence, the sooner we drop the word "fever" as indicative of anything specific in any given disease, the better it will be for our reputation as philosophical thinkers and writers.

In this sense, then, I propose that this disease be hereafter spoken of as the Southern Cattle Plague of the United States.

*Literature: Transactions of the New York State Agricultural Society, 1867, Vol. 2. Reports of the Department of Agriculture: Diseases of Cattle in the U. S., 1871; do., 1880-81, 1883, 1884, 1885.

DEFINITION.

Although we shall have to refer to this subject in detail in considering the nature of the southern cattle plague, still it may be well to notice it briefly at this point.

First, we will consider the ideas of our predecessors in this field of investigation.

Gamgee says: "This splenic or periodic fever, commonly known as Texas fever, Spanish fever, or cattle fever, is a disease peculiar to the ox tribe, which has never been described as attacking southern cattle, and which occurs in a more or less latent form among them. It is, so far as we have ascertained, incapable of communication by the simple contact of sick with healthy animals; and in the strict sense of the terms is neither contagious nor infectious." Report 1871, p. 84.

More illogical and absurd contradictions than the above could not possibly emanate from the merest novice upon disease.

If any disease "occurs in a more or less latent form," it certainly cannot be described as "not attacking" any class of animals; the very statement that it "occurs in a latent form" is admitting that it does occur. There is scarcely a contagious or infectious disease afflicting animal life which does not occasionally occur in such a "latent" form, the reasons for which in many cases are beyond our knowledge, but plain enough to be seen in regard to the southern cattle plague, as will be shown elsewhere.

Again, Gamgee tells us that "in the strict sense of the terms it is neither contagious nor infectious." Then what in the name of common sense and logical reasoning is it?

Immediately after saying that, he says: "It is an enzoötic disorder."

We would ask any student in medicine if he did not understand that by a disease being an "enzoötic disorder," it is either a contagious or infectious disease, the outbreak of which is limited to a small extent of territory?

But Gamgee himself gives such positive testimony that the disease "in the strictest sense of the terms" must be either contagious or infectious, that one is at a complete loss to know what the man was thinking about when he wrote that "it is neither contagious nor infectious."

In one place he quotes a gentleman as saying:

"Talk to a Missourian about moderation when a drove of Texas cattle is coming, and he will call you a fool, while he coolly loads his gun and joins his neighbor; and they intend no scare, either." Report 1871, p. 82.

"In Texas, cattle of all ages are afflicted with the malady in a somewhat latent and mild form." Ibid., p. 25.

"The period of incubation is generally five or six weeks." Ibid.

"Animals recover, especially in the South; but the communicated disorder among northern stock is very fatal." Ibid.

"It is evident that a large herd, traveling from the region where Texas fever is propagated, carries not only the active cause of such propagation in the systems of the animals composing it, but the evidence of a specific disease induced, which remains for an indefinite time latent and unobserved." Ibid., p. 87.

"It is proved that the animals [southern cattle] may simply pass leisurely over a road or prairie, feeding as they move along, and without remaining for any length of time on any portion of the ground they traverse, leave behind them sufficient poison to destroy all or nearly all the cattle [northern] which continue to feed upon it." Ibid., p. 88.

Further quotations need not be made to show that Mr. John Gamgee did not know what he was writing about when he said that this disease is "neither contagious nor infectious," for everybody who knows anything, knows that diseases which have a definite period of incubation — that diseases which call men out with shot-guns to prevent losses in their own stock — that diseases which are transported over a country by individuals, whether in a latent or manifestly diseased form — must of necessity be either contagious or infectious in the very strictest sense.

Mr. D. E. Salmon does not exactly tell us what he considers this disease to be; but he is certainly as full of contradictions and uncertainty as to its nature as Mr. Gamgee. He says:

"It was only contracted from infected grounds; sick animals seldom if ever spread the contagion; a fence was sufficient to arrest the disease." Report 1883, p. 29.

Who ever heard of a contagious disease acting in any such manner as that? Who ever heard that animals sick with a contagious disease "seldom if ever spread the contagion," when the fact is that that is the chief way by which contagious diseases are spread?

As will be shown in future remarks, the only logical conclusion as to this disease is, that it is an *Extra-Organismal-Infectious-Septicæmia.*

Geographical Distribution of the Southern Cattle Plague.

So far as is known to us in the United States, this bovine pest is peculiarly an American disease. It is more: it is a disease which finds its original development in a limited portion of the United States, and especially in those parts of our southern States (and the lowland portions of the same) which border upon the Gulf of Mexico on the one hand, and our Southern Atlantic ocean on the other. Whether or not the disease exists in similar localities in Mexico, Central and South America, that it is in the lowlands of those states bordering on the sea coast, is a matter upon which no data can be found; but it would seem natural that if northern cattle were imported into those countries, they would be liable to encounter the same danger.

The disease is not known to exist in any part of Europe, Asia, or Africa, but that is not saying it does not. There has lately appeared a short description of a disease among cattle in Italy which is of sufficiently suggestive value to warrant its introduction here, as it looks very much as if the disease mentioned were this plague, or a near relative. It is to be found in the "Veterinary Journal," (London,) Vol. XXV., (1887,) p. 422, and is entitled, "Study of Bacillus Causing Nephritis in the Ox," by Professor S. Rivolta:

"This disease has been observed as an enzoötic in the Roman Campagna during the summer, particularly in pure-bred Swiss and Dutch cows that have been recently imported.

"Professor Vogliata recognized this disease as what has been described by Metoxa as 'ataxo-dynamic nervous fever,' by others as 'pernicious fever' and 'hæmatura,' with the following symptoms:

"At the first onset, elevation of temperature to 39–40° C., the urine turning to a dark red color, with a greenish tinge through it. The animal becomes dull, the coat stands upon end, the eyes are sunken, the mucous membrane tinged with yellow, and the fæces hard. Motion is difficult, the hind limbs being moved with difficulty. A little later rumination ceases, and the temperature rises to 41° C. The urine becomes coffee colored, and muscular spasms set in, the pulse being very quick. The temperature then rises even higher, and then descends, the animal dying without convulsions.

"After death the blood is found black and fluid, as in anthrax, the liver yellow and engorged with blood; the spleen tumefied and nodulated on its surface, and the bladder distended with bloody urine. The lungs show sub-pleural hemorrhagic patches, and the endocardium petechiæ."

Before giving the remarks upon the micro-organism found by Rivolta, it may be well to call attention to the resemblances between this disease and the southern cattle plague of the United States.

Whether the fault of the translator or the original observer, it will be seen that the necroscopical notes are very meagre indeed. In fact, though described as a "nephritis," still we have no description whatever of the appearance of and lesions in the kidneys. On the other hand, here we have a disease apparently peculiar to cattle, occurring in about the same latitude and under about the same telluric conditions, and above all in imported cattle from a northern latitude, exactly as occurs in the American cattle pestilence; but on the other hand, we miss any allusion to the deportment of the cattle native to the district in which the disease appears as an enzoötic. Again, while there is nothing specific or pathognomonic in the symptoms of this disease, as described, any more than there is in the southern cattle plague of this country, (for the bloody urine is not peculiar to either, occurring as it does in anthrax and from other causes,) still the symptoms described could answer as well for the American as the Italian pestilence. The same is true as to the pathological lesions quoted, especially as to the condition of the liver, which bears the greatest resemblance to that organ in southern cattle plague, as well as the spleen, which is not pathognomonic to either disease. It would have been of great comparative value had the condition of the intestines and lymph-glands been also mentioned. The constipation noted is also common in the cattle plague of this country.

As to the micro-organism discovered by Rivolta, the description is also so meagre, in the article at command, as to be almost valueless for comparison, as all it amounts to is the assertion that a "short fine bacillus" was found, but no description of the morphological or biological phenomena of the germ is given. However, we will let the original remarks follow:

"Professor Rivolta discovered a short fine bacillus in the blood, differing from that of charbon (anthrax) under the microscope, but not in preparations made with the pulp of the spleen. (? B.) Sections of the kidney showed sub-acute interstitial nephritis, as well as the bacillus found in the blood, which was demonstrated by Gramm's method. They were present isolated, and united in twos and threes, straight, curved, and bent at an angle, with an imperfect segmentation. Their length varied from $\frac{2}{1000}$ to $\frac{5}{1000}$ and their breadth $\frac{1}{1000}$ of a millimeter.

These bacilli were never found in the spleen or other parenchymatous glands, although their presence in the blood would indicate their existence in these organs."

"Inoculation on the rabbit and pig had no results; neither had they in those that were made on cattle; and the author arrives at the conclusion that the results are only obtained in animals previously disposed."

[Rabbits and hogs are immune to the action of the southern cattle plague also.—B.]

The conclusions Rivolta arrived at are:

"1. That a sub-acute interstitial nephritis exists in cattle, due to a specific bacillus.

"2. That this disease, with its accompanying hæmaturia and hœmoglobinuria, causes obstruction in the Omasum. (!!!? B.]

"3. Many bacilli colored with gentian violet show their protoplasm in a small round mass."

[I take this to mean something corresponding to the pole-ends of the germs of our southern cattle plague, and that the imperfect segmentation may have reference to the "belted" appearance caused by the non-coloring substance. This is all an hypothesis, waiting further confirmation.—B.]

"4. In the kidneys they multiply by division, and are transported by means of the lymphatics and blood vessels.

"5. The majority of the short bacilli appear to have their habitat in the lymphatics."

[Above it has been said "they never are found in the spleen or other parenchymatous organs." We would ask what are lymph-glands but "parenchymatous organs?"—B.]

"6. The bacillus, from its irritative action on the kidneys, Rivolta has named the "bacillus nephritis bovis."

[A most improper nomenclature, as it gives us no idea of the true nature of the disease, and even from the description given it is evident that the renal lesions are not the essential ones of the disease, but rather those of the blood, and that the disease is also a septicæmia. —B.]

Some European authorities have been led to think that the American disease was the European rinderpest; but that question does not need to be argued, as that disease is not a climatic disease; nor does it die out under the influence of protracted freezing; nor do fences keep

it off from susceptible cattle. In fact, the rinderpest is an endogenous while our southern cattle plague is an exogenous disease in *optima forma.*

This disease again assumes a peculiar interest from the standpoint of comparative etio-pathology, in that it occurs in the United States in the same latitudes, and under almost if not the same climatic and telluric conditions, as does the yellow fever in man, and in many other ways bears a very close resemblance to it.

Being a disease, the primary development of which is dependent upon certain known or ascertainable climatic and telluric conditions, it is essentially a local disease, and in general these dangerous parts of our country have become pretty well known. It is, however, very evident that to our better knowledge of its origin, and to elucidate more conformable regulations for the movement of Southern cattle and the prevention of the disease in the North, the most exact and detailed study of such localities is an obligation resting not only upon our general government, but also upon those of our Southern States, in order that the greatest latitude possible with safety may exist in connection with the development of our live stock (bovine) interests, especially as they bear the closest relations to our public health as the means of supply for our chief (or perhaps better, favorite) article of animal food.

As has been shown in the brief historical sketch previously given, our Agricultural Department at Washington made a very good beginning in the study of this disease in the years 1869 and 1870; but while some attention has been given to it since, still it has not received the notice that its importance deserves, though some new evidence has been gained as to its extent over the country. It is to the disgrace of the governments of those states, especially that of Texas, in which it primarily originates, that no proper steps have ever been taken toward its scientific investigation, important as the disease is to the grazing interests of those states which are largely dependent upon the Northern States for a market for their food-producing animals. In fact, as will be shown, it has been and is still largely the policy of those states to deny not only the existence of the southern cattle plague in their cattle, but even to deny the possibility of the outbreaks of any such disease in northern cattle being in any way connected with cattle from their respective states, especially Texas;

notwithstanding the immense amount of costly experiences of the most positive and incontrovertible character, which northern cattle growers have undergone from year to year for a long series of years.

The later work of the United States Department of Agriculture has been in the direction of fixing the amount of territory covered by this disease in its permanent and original development.

From the annual report of that department for the year 1883, the following quotations have been taken:

"The permanent home of Texas fever was formerly believed to be confined to the Atlantic coast, south of North Carolina, and the Gulf coast from Florida to Texas. Having in former reports given many facts tending to show that a part of Virginia, nearly all of North Carolina east of the Blue Ridge, and the greater parts of the States of Georgia, Alabama, Mississippi, and Louisiana were long since overrun with this plague, it is proposed to call it the southern cattle fever." Page 19.

Evidence seems also to have been collected tending to show that the

TERRITORY OF PERMANENT INFECTION

is gradually extending in a northerly direction, as may be seen from the succeeding remarks:

"It has been demonstrated, beyond any possibility of successful contradiction, that the district permanently infected by Texas fever is being continually enlarged by the advance of the infection towards the north. Page 31.

Again, this report says upon the "*Rate of advance of the infected district:*"

"In collecting other facts with regard to Texas fever, we have endeavored to obtain data which would enable us to determine the rapidity with which it is advancing, and the time which it will require, in the future, to gain a certain distance. The evidence bearing upon this point is still very insufficient, but it is not without considerable value. It certainly gives us a more definite idea of the matter than we ever had before. The most rapid progress, for a long series of years, has been made in North Carolina in the extension of the disease from east to west."

"About fifty years ago, as would appear from the laws enacted at that time, the border line of the infected district was somewhat east of Raleigh, where the character of the timber changes, and the long-leafed pine appears. This line is now at the Blue Ridge mountains, a distance of at least two hundred miles, or average of four miles a year."

"In Habersham county, Georgia, there appears to have been an advance of about twenty miles in the course of ten years. In Franklin county, Virginia, it has advanced twelve miles in three years, or four miles a year. In certain parts of Henry county, Va., the progress has been rather over three miles a year for the past two or three years. In Halifax county it does not seem to have advanced more than twenty miles in sixty years. In Campbell county the advance has been but ten miles in fifty years; while in Buckingham county there seems to have been scarcely any new territory covered for the last fifty years." Pages 42 and 43.

Accepting these statements as approximately correct, they show a most valuable line of research which should be kept up annually, not only by our National Agricultural Department, but by that of every state in the suspicious parts of the country. This is especially true of the Southwestern States and Territories which are depended upon to supply a large proportion of the feeding cattle for the more northwest and corn-growing states. In this regard there is far too much laxity on the part of the respective state governments. They seem to assume that it is all right for them to leave this kind of work to the National Agricultural Department, and do nothing themselves; whereas each state should do all it possibly can, and act as if the welfare of the whole country depended upon the character of the investigating work done within its own borders.

But little of value is ever done, where everybody waits for some one else to begin to do it, as has been the case with regard to the study of our animal diseases and taking proper measures looking to the better protection of our great live-stock interests.

EXTENSION IN SOME OTHER STATES.

Tennessee.

According to the Report of the Department of Agriculture for 1884, the State of Tennessee is also gradually becoming permanently infected, the disease extending from the southern toward the northern part of the State. "Even the mountainous countries in the southeastern part of the State have been invaded."

Kansas.

Hon T. J. Shepler reports:

"That a prominent stockman from Montana assured him, last spring, that cattle coming from Kansas would transmit the Texas

fever to their cattle in the most malignant and fatal form." Report 1884, p. 409.

Arkansas.

"It would appear that the northern line of infection leaves the Mississippi river at or about the southern boundary line of Mississippi county. This is very nearly opposite the point in Tennessee, on the Mississippi river, to which the boundary line of the infected district was traced in that State. Going westward, this line would appear to follow the southern boundary line of Poinsett county, crossing Jackson and Independent counties, going a few miles north to Batesville, and then proceeding westward through Stone and Searcy counties, leaving Mountain view and Marshall a few miles to the south. It then takes a more decided northwestern direction, crossing Newton and Carroll counties to Eureka, and is then directed westward to Bentonville, and from this town goes to the extreme northwest corner of the State." Report 1885, p. 248.

Indian Territory.

"Sufficient observations of a definite nature have been recorded, however, to show that the line of infection continues in a northwesterly direction from the northwest corner of the State of Arkansas until it reaches to within twelve or fifteen miles of the Kansas State line, at a point south of Chetopa. Its direction is then westward across the Cherokee country and nearly half-way across the Osage country. It then takes a southwesterly direction to the Texas line, crossing the country of the Kiowas, Comanches, and Apaches." Report 1885, p. 249.

Texas.

"Different organizations of stockmen and different individuals have different ideas as to the location of the infected districts in this State. It is evident that a definite line cannot be traced from such information as this. The only way to draw a definite and safe line of demarkation is to consider the definite observations of stockmen as to the effect of moving cattle from one given part of the State or of the country to another given point. When a large number of such observations are collected, then we have reliable data upon which to found an opinion that cannot but be in accordance with the fact. Believing this to be true, a line has been drawn from the Red to the Rio Grande rivers in such a position that it may be assumed that all the country west and northwest of that line is free from any permanent infection, and the native cattle from it may be safely taken to any part of the country without disseminating the inficiens of southern fever. This line must therefore be considered as a preliminary line, based upon such positive information as has been possible to collect, and is

subject to revision and change according to investigations which may be made in the future.

"The counties most likely to be uninfected, according to leading stockmen of Texas, are Shackelford, Callahan, Taylor, Runnels, Coleman, Poncho, McCulloch, San Saba, Mason, Llano, Gillespie, and perhaps others which are of a more or less mountainous character." Ibid., p. 273.

It has been previously said that many Texas cattle men deny the existence of any such disease as the so-called Texas fever among native stock. A few examples of their assertion may not be uninteresting. One party writes:

Our opinion is, that the Texas fever is unknown in our native Texas cattle, and we do not believe they are capable of imparting a disease they do not have." Ibid., p. 255.

Others contradict the above assertion.

A gentleman from Gainsville, Texas, says:

"There have been no cattle turned in our range from southern or southwestern Texas, but will say that we lost cattle in 1883 from driving on the trail behind southern Texas cattle, and from contact with them, with the disease known as Texas fever."

Another:

"Will not allow cattle (southern Texas) to mix with his herd without a thirty days' quarantine of all cattle from a lower altitude than 1000 feet above sea level. So far as my observation has gone, Texas fever is confined to cattle in the extreme southern part of the State." Ibid., p. 259.

With regard to Texas fever in Texas, I have a very interesting letter from a veterinarian whom I know well, and in whom I have much confidence. Under date of September 12, 1887, he writes:

"We have here in Texas the same disease that you call 'Texas fever' in the North; though many of the so-called veterinarians say that 'there is no such thing here.' That is nonsense! We have also much anthrax, but the cattle men do not seem to know it. The cow and horse doctors are mostly Mexicans, or cow boys, who perform the most wonderful cures if one could believe half that they tell. I have seen much Texas fever within a few months; the cattle men call it 'milt fever' in the prairie districts, and the same disease on the highlands is known as 'mountain fever.' The ignorance and confusion with regard to animal diseases is simply horrible in this part of the country."

HISTORY.

The historical extension of any such devastating animal disease as the Texas or southern cattle plague of the United States cannot be without interest to every breeder and owner of bovine animals, but also to the entire veterinary profession of the world. All that is at present known about this disease is to be culled from the various reports of the United States Department of Agriculture, and it is to them that we must turn for information connected with its history and extension. Our first knowledge of the subject is to be gained from the report of 1871, and as no other work refers to the part this disease has played in the historical development of our country, the writer must be pardoned if he offers a verbatim copy of the same from that report, especially as there is not sufficient material of the same nature in the later reports which would justify the labor of attempting a historical compilation of the facts. This is all the more pardonable as the report in question is very rare and almost "out of print."

"Two years prior to the initiation of the series of investigations chronicled in the preceding pages, (of this report,) and long before the public mind of the Atlantic States was aroused to the dangers of the summer transportation of cattle fresh from the plains of the Gulf States, there was undertaken a systematic investigation of the facts stated and reiterated by reliable farmers in the track of Texas-cattle migration, stoutly denied by Texans, referred by drovers to every cause but their own cattle, and faintly believed or mildly doubted by the people, and even by the papers of the East. Some affected to regard the reports from Kentucky, Missouri, and Kansas, as mild exaggerations of the truth, or fabrications in extenuation of controversies and violence begotten of encroachments upon the ranges of the cattle growers of the border. But the reports were too general and the statements too direct and fortified by substantiations too strong to be wholly ignored. Besides, they have been repeated year after year since the introduction of southern cattle in the North, not only in those states, but in the more eastern states of similar latitudes or climatic conditions. The drovers of Florida and Georgia, in the past generation, had witnessed similar results from the movement of coast cattle; and indeed the disease characterized in the preceding report as Texas fever can be distinctly traced back into the eighteenth century.

"It has been in existence ever since cattle were first driven from the country bordering on the coast of the Mexican gulf to the upland

regions to the northward, wherever cattle were present on the line of march to receive the infection.*

"The existence of this disease, proven by adequate testimony from many places and through a long period of time, was still either positively unknown or practically ignored by agriculturists at a distance from the places of its prevalence; so that, on the introduction of Texas and Cherokee cattle, through the swift intervention of steam, by river and by rail into the heart of the Ohio valley, the results hitherto invariably occurring among Kansas or Missouri stock now visited with equal severity the cattle of Illinois and Indiana, and forthwith the doubt and indifference with which a distant calamity was regarded were exchanged for apprehension and alarm, which spread rapidly eastward, awakening the anxiety of stock owners, arousing to action city boards of health, and causing panic among purchasers of meats. Even agricultural editors, ignorant of the real character of the disease, wrote of the probabilities of its dissemination from farm to farm like the virus of rinderpest, a result of which no fears could have been reasonably entertained, native stock having the disease not communicating it to others."†

By referring to the writer's observations, it will be seen that the above statement is incorrect, and that both Dr. Rauch and the metropolitan board of health of New York had cited cases of this kind (1867) long before this report was published, (1871,) though, as will be shown, their conclusions must have been erroneous. It was left for us in Nebraska, to observe the first undoubted case of this kind, and to prove it beyond a doubt, as well as to show why such secondary infection had not previously occurred in the history of the disease. Were this not so, the southern cattle plague of our country would be an unheard-of anomaly among the diseases of the world; but, given time and favorable climatic and telluric conditions, this disease will as certainly be extended through northern cattle to other northern

* The above remarks have no reference to the history of this disease in its native clime, but only to the period when attention was first called to its extension to other localities by means of cattle from its native heaths. It is highly probable that its specific cause has existed in our southern states for centuries before we had any idea of its being the cause of a specific disease in cattle. The same is true of the yellow fever. If the Indians, prior to the English settlement of the states, ever brought with them in their northern migrations Spanish or Mexican cattle which were native to or had been acclimated in the lowlands, it is more than probable that this disease was communicated to the cattle of the higher latitudes.

† To this remark is added the following foot note: "No fact connected with the Texas cattle disease is more firmly established than this: Among all the records of its ravages in all the years of its history, no instance of a secondary generation of the virus, no statement of its communication from a sick northern animal to a well one is noted, with a single exception, which, if really an exception at all, only serves to establish the rule."

cattle, as swine plague to swine, or anthrax to susceptible animals grazing on infected lands, and with equal certainty to the infection of northern cattle when transported to the native heaths of this disease in our Southern States.

"Yet this alarm, notwithstanding the extravagance of its manifestation, accomplished good results, calling public attention to the abuses in cattle transportation, exciting inquiry which resulted in more intelligent views of the subject, aud promoting legislative action protective of the stock-growing interests.

"The first notice of this disease which we have been enabled to find is in a lecture delivered before the Philadelphia Society for Promoting Agriculture, by Dr. James Mease, Nov. 3, 1814, upon the diseases of the domestic animals, in which it is stated that cattle of a certain district in South Carolina 'so certainly disease all others with which they mix in their progress to the north, that they are prohibited by the people of Virginia from passing through the State.' It was mentioned as a singular fact that the South Carolina cattle had the power of infecting others with which they associated, while they themselves were in perfect health; and also that cattle from Europe or the interior brought to the vicinity of the sea were attacked with a disease which generally proved fatal. Dr. Mease corroborates these views from personal observation in Pennsylvania in 1796. Sept. 20, 1825, he read before the same society an 'Account of a Contagious Disease Propagated by a Drove of Southern Cattle in Perfect Health.'"

The following extract is given:

"In the month of August, 1796, I was on a tour for the recovery of my health, and having called at Anderson's ferry, now Marietta, in Lancaster county, on the Susquehanna, I found the people of the house in great distress on account of the death of some cattle and sickness of others which had occurred in a few days after a drove from the South had left the place. Upon inquiry, I was informed that the drover merely requested permission to confine his cattle for one night in a plowed field, and I was assured that the stock of Mr. Anderson had no intercourse with the drove, which, after staying all night, pursued their journey in the morning to Lancaster. There several head were disposed of to different persons, and in every instance I was informed that they communicated disease to the stock with which they mixed. The admission of a single head was enough to give rise to it. As the drove of cattle exhibited no mark of disease, the mystery of the case was inexplicable, and is to this day. They stopped for a day or two at Downingtown, thirty-two miles from Philadelphia, and soon after the field they occupied received another drove, which consisted of two hundred and sixty head which had been

purchased in Maryland. Sixty of this drove were sold near Billet, Montgomery county, the great part of which died, and others sold in different places also died. Part of the South Carolina drove were sold at Blue Bell tavern, a well-known sale place for cattle, and of these forty-six head were purchased by Mr. ———, who then rented the meadows on State Island, and were mixed with about two hundred and seventy others. In about four days after the southern cattle had been turned out on the meadows, they were brought up to the yard round the barn to be branded, when they were returned to the pasture. The disease first appeared after a few days, among the cows in a field near the barn, and which were regularly milked in the yard used to confine the southern cattle until branded, and in a pair of fine work oxen, which were regularly and daily fed and yoked in the same yard. Several other cattle were successively attacked, to the number of at least twenty; all of them, except one, died. All those purchased in June died. My advice being asked, I went to the field where several cattle lay ill, and was told that the first symptoms were loss of appetite and weakness of the limbs, amounting to inability to stand. When they fell they would tremble and groan violently. I saw several in this condition. Some discharged bloody urine. The bowels were generally very costive. Upon being opened, the kidneys were found inflamed and sometimes in a state of suppuration, and intestines filled with hard balls. The blood was in a state of decomposition, and did not coagulate. None of the southern cattle died. The circumstance of the cattle from a certain district in South Carolina infecting others with the disease above alluded to, has been long known; but the precise locality, or its extent, I have not yet been able to ascertain. The country of the long-leafed pine is said to be the native place of the infection, but with what certainty I am unable to say.

"Old residents of the Piedmont region, between the tide-water areas and the Blue Ridge, are familiar with this disease, and the cattle drovers who have brought stock from the country of the long-leafed pine to a greater elevation and higher latitudes, testify with remarkable unity to the constancy of its appearance and the uniformity of its prominent characteristics.

"The following statement, obtained by the statistical division of the Department of Agriculture, April, 1867, from Mr. J. Wilkinson, of Athens, Georgia, a reliable cattle dealer of good judgment and great experience, embodies the essential points of this oft-repeated testimony:

"'I have been a cattle dealer for about thirty years, and in that time have had many deaths among my stock by this disease, and have, in consequence, taken some notice, meanwhile endeavoring to learn its causes and how it was brought about. I notice that cattle scarcely

ever take the fever if let remain where they are raised, and I am fully convinced it is generally brought about by change of climate. For instance, you take cattle from the mountain country to the low country, and they will take the fever in a short time and die; but their disease will not affect the cattle raised there; but, on the other hand, take cattle raised in what we call the distempered part of the country — that is, the low country — from warm latitudes up into a colder one, they will improve all the time; but, without being sick themselves, they will spread the fever and kill the cattle in the section of country into which they are taken, or have stayed long enough for the fever to leave the system. How it is that they carry the disease with them and give it to others without injury to themselves, is a mystery I am not able to solve, and will leave that to be discussed by the Bureau of Investigation."

During the war of the Rebellion, as there was no commercial intercourse between the Northern and Southern States, it was but natural that there were no outbreaks of the southern cattle plague in the Northern States, as no southern cattle were imported. Hence it was not until the year 1867 that any new outbreaks were chronicled. Quite extensive eruptions then occurred, on account of the great number of importations, the cattle being very cheap in the south, especially in Texas.

"The Agricultural Department of Washington, as well as several local boards referred to in other parts of this treatise, again began to give serious attention to this disease, and the results of their investigations demonstrated the truth of previous information and the traditions of the earlier cattle trade in the south, showing that the disease has hitherto only developed among natives (that is, northern cattle) upon the importation or arrival of Texas cattle in southern Kansas and Missouri, and the more elevated sections of Arkansas, in parts of Tennessee, southern Kentucky, North Carolina, and the hill lands of Georgia and South Carolina. It was not reported farther north than southern Illinois, and its existence seemed to be then unknown in Ohio, Pennsylvania, and Maryland. A fact suggestive of its climatic origin showed its existence in the mountain lands of Georgia, where it was generated by the presence of lowland cattle that had scarcely been removed a distance of fifty miles. It appeared that cattle driven from Texas to New Orleans did not communicate the disease to Louisiana cattle. Nor was there any evidence that the cattle of any lowland section, when driven to another, caused an outbreak of the disease. A marked instance was reported from Arkansas, eight hundred cattle having been driven directly from Texas into Mississippi county, in 1866, where they remained and mingled safely with the

native stock. This country lies in a latitude sufficiently high to awaken expectation of a fatal result in such migration; but it is not on the Mississippi, in a miasmatic region."

The writer of the above fails to appreciate the fact that all Texas cattle are not the bearers of the inficiens of this disease, as all parts of Texas are not by any means dangerous, and the cattle, in the above case, may have come from a part of Texas where the disease does not prevail, the climatic conditions not offering favorable factors thereto.

The Texas correspondents (of our Agricultural Department) were indignant in their comments on the Texas cattle fever. Many claimed that their cattle were not subject to any prevailing disease. One, in Collins county, admitted that cattle brought there from the North are liable to a disease similar in its symptoms.

"There is no doubt that the cattle from Texas are thrifty and comparatively (or better, apparently) free from disease, but post-mortem examinations in Texas and at the abattoirs of northern cities, show enlargement of the spleen and traces of former derangements of the digestive organs. It is also a fact that the annual reports upon the condition of live stock to the Agricultural Department, from the miasmatic sections of the country, contain accounts of fatal "murrains" more frequently than those from elevated locations and higher latitudes. 'Murrain' was reported, in the returns of the spring of 1867, in many portions of the South, without any details of the symptoms or circumstances, but in many cases with descriptions highly suggestive of the southern cattle plague; such as 'cattle pastured with cattle from the South take the murrain and invariably die, though those brought from the South do well.' The investigations of 1867 showed that the movement of Texas cattle north, which had ceased during the war, was being again prosecuted with vigor, bringing with it the old disease, which raged just in proportion to the extent of the movement of southern droves. Its ravages in 1866 were mainly confined to Kansas and Missouri, with a few instances of its prevalence in Kentucky and Southern Illinois."

In 1868 the disease broke out again in the North, and became much more rapidly extended as the cattle from the South began to be shipped by rail. It spread rapidly through Indiana and Illinois, and even isolated but alarming outbreaks occurred in Ohio, New York, New Jersey, and as far east as New England, being in every case caused by the shipment of southern (in general Texas) cattle to those localities. It was at this time that our Agricultural Department availed itself of the services of the then eminent veterinarian, Mr.

John Gamgee, of England, who was in the country at the time, and whose investigations upon this bovine scourge are among the best that we have.

The investigations of our Government up to this time had led to the following conclusions, several of which are erroneous:

"1. That the disease is communicated (to northern cattle) by cattle from Texas, Florida, or other states bordering on the Gulf of Mexico.

"2. That the disease itself is unknown in Texas."

[Here it should have been said, denied by Texans to exist in their native stock, but still attacking them in a mild form, and as dangerous to northern cattle taken to the infected portions of Texas, as Texas cattle are to northern cattle when driven north, through their ability to infect our northern lands.]

"3. That the (southern) cattle communicating it are not only apparently healthy, but generally improving in condition.

"4. That while local (northern) herds receiving the infection nearly all die, they never communicate the disease to others."

[A mistaken conclusion, as will be shown later on.]

"5. That either a considerable increase in elevation, or a distance of two or three degrees of latitude from the starting point, (of southern cattle,) is necessary to develop the virus into activity and virulency; and a further progress of two degrees of latitude, or a few weeks in time, is sufficient to eliminate the poison from the system" (of southern cattle).

The above is incorrect, except the last remark, which simply states a fact.

The inficiens of the southern cattle plague does not increase in virulence, or "develop into activity," in the organisms of southern cattle. On the contrary, it really loses in virulence in passing through the bovine organism, notwithstanding the great susceptibility of the same to its action. It possesses its full activity and virulence in Texas, or a southern clime, where it finds its original development and retains the same, or even acquires it again when planted on our northern pastures by Texas or southern cattle, when the climatic conditions are the same as those of Texas. A more illogical or unpathological statement — in fact, a more nonsensical assertion — could not be made than "that either a considerable increase in elevation, or a distance of two

or three degrees of latitude is necessary to develop the virus into activity and virulence." In fact, this assertion is absolutely contradicted by the next one, "that a further progress (of the cattle) of two degrees of latitude is sufficient to eliminate the poison from their systems." According to the first assertion it increases in virulence in their systems, while according to the second it loses its activity altogether in "a few weeks in time."

[As I will show later on, the southern cattle are simply the conveyers of the inficiens from southern to northern lands, and when their intestines become entirely freed from the herbage and water taken up on their native lands, they are then unable to cause any infection of our northern pastures, and hence, our cattle.]

"6. That Texas cattle removed to other miasmatic sections, as the Mississippi bottoms up to the thirty-sixth parallel, communicate no infection to local herds."

[Which simply shows that the inficiens finds its home there and equally favorable conditions to its development to those offered by the plains of southern Texas.]

"7. Medication is useless!"

This is about all we know of the history of the southern cattle plague of the United States, except such as will be noticed in the text in the appropriate places.

Since 1868 the different states have had more or less stringent regulations prohibiting the importation and movement of southern — more especially Texas — cattle over or into their territory. For awhile these regulations restricted the traffic in these cattle; but as the execution of the laws bore no relation to their severe tenor, and as the inspection of such cattle is a mere farce, the trade has of late increased, and in consequence numerous outbreaks of Texas fever have appeared in a large number of our northern states.

Part II.—Etiology.

THE ETIOLOGY OF THE SOUTHERN CATTLE PLAGUE.

No disease that occurs in animal life has but one cause. This axiomatic fact applies equally as well to endogenous and exogenous diseases as to others. In order that we may be able to prevent a given disease, it is absolutely necessary that we first arrive at a correct conclusion as to its exact and true nature and causes. Without this knowledge, all attempts at its radical prevention will prove futile. A knowledge of the true nature of an infectious disease is of far more importance in considering means to prevent it than a knowledge of its specific cause. The same is also true as to the character of the secondary or supporting causes, without which the specific cause could not act.

These causes may be classed as:

Internal—that is, specific-racial, or constitutional;

External, or supporting; and

Specific, or exciting causes—the inficiens proper.

With regard to the first or internal causes, that is, that peculiar condition of the constitution of a given species of animal life which predisposes it to the action of the specific or exciting cause, the *causa sufficiens* of infectious diseases, we know but very little more than that the fact exists, and that it is an hereditary constitutional attribute peculiar to animal life. We know that no matter how healthy an individual may be, if it, or he, is exposed to the action of the specific cause in any of the two classes of infectious diseases, that individual will generally become infected, inoculative or acquired immunity always excepted. This is true of human beings with regard to the small-pox, scarlet, mumps, etc.; glanders in horses, the pleuropneumonia in cattle, the swine plague in swine, and the southern cattle plague in the bovine species. There are other diseases, however, where this predisposition may exist in a large class of animals; but where it is not at all a predisposing attribute in any such sense as the above. Such diseases can only be transmitted from one animal to another by

direct inoculation, artificial or accidental. Rabies is the most striking example of this class. There are also animals which have no constitutional predisposition whatever to a certain disease of this class, and it cannot be induced in them in any way we may try. The relation of cattle to glanders illustrates this fact. Glanders cannot be produced in cattle. Syphilis is limited to the human species.

The external or supporting causes are many; they include anything and everything that takes or may take part in extending a given disease from one animal to another, or from one place to another, or in supporting the life of the causa sufficiens outside the animal organism. With regard to endogenous diseases, such causes are known as "vehicles," or conveyers, of the infecting principle. They cannot support its life, but they can be the means of its being conveyed from one animal to another, or even from one place to another. They are either something polluted by material from a diseased animal or some part of such an animal. In exogenous diseases the same thing may occur. In this class the diseased animal is an external cause with relation to other susceptible animals. It is also a vehicle of infection between two different localities. These external causes are of far more importance in the consideration of exogenous than of endogenous diseases. We shall consider many of them very fully in the treatment of the nature of this disease, but their importance makes it necessary that they receive some consideration in this connection. Among the most important of them, in purely exogenous diseases, are the conditions of the climate and soil; because, unless these are favorable, the *causa sufficiens* is without means of active existence. It is because of them that this cause retains its vital activities for an indefinite period; and it is because they play so unessential a role in endogenous diseases that the same cause retains its vitality but a limited time outside of the animal organism. It is because of this peculiar relation of these external causes to the *causa sufficiens* that the prevention of the strictly endogenous diseases is a comparatively easy matter when slaughter and disinfection or — really better — thorough cleansing are rigidly applied, while the same procedures are of but little avail in the prevention of the exogenous diseases of the character we are considering. Even here cleanliness is of far more importance than disinfection. These external causes — that is, climatic and telluric conditions — are not always necessary to the preservation of the vital activity of the germs of cer-

tain exogenous diseases. This remark, however, only applies to those which develop permanent spores, a biological condition in which it seems as if certain germs could retain their vitality forever.

Anthrax gives the most striking example of this fact. We have in the laboratory some pieces of string that I dipped in a fluid culture of *Bacillus anthracis* in 1878, when working on this disease in the Bavarian mountains with Professor Feser, of the Munich Veterinary School. It is now over ten years since these were obtained, and they are as infectious as ever. They have been kept absolutely dry in a glass vial ever since, and hence without anything to support or disturb their vitality.

But, aside from those already mentioned, we have other external causes which act as vehicles of infection in *exogenous* diseases. These are the excretions of animals, particularly the manure and urine, that is, media by which the inficiens proper is again sowed upon the land. Aside from these, we have to consider the animals which have perished from the disease. Now all these factors constitute the external or supporting causes, among which we must not neglect the water courses and conveyances of common carriers, all of which may, and not unfrequently do, play a very essential role in the extension of exogenous diseases over the country. They have less importance in the consideration of the majority of endogenous animal diseases, especially the water ways; but cars, omnibuses, etc., are not without serious effects in the extention of such diseases.

The specific cause, the *causa sufficiens*, is also an external cause, but of an entirely different nature. A knowledge of the exact character of the external supporting causes is far more essential to the proper understanding of a disease than is that of its specific cause. In fact, one might send a cultivating tube containing a culture of the germ of any given disease to a botanist, and he might tell us all about the morphology and much about the biology of that germ. In this sense he might even exceed us in skillfulness, but his knowledge would be of no value in the prevention of the disease, simply because he knew nothing of the disease itself and its supporting or external causes. It is only when we take the latter into full consideration that our knowledge of bacteriology has any practical value in disease. It is his pathological acumen, and not his cleverness as a technician in bacteriology, that makes the pathobacteriologist of value to his race. It

is high time that the medical profession as well as the public began to appreciate this fact. Bacteriology has been passing through its "fashion" period in medicine. Men have been by far too anxious to have their names connected with the first discovery of *a* germ, rather than *the* germ in connection with any special disease for the best reputation of medical research.

Too much attention has been given to hunting adventitious germs, for the credit of original investigation. The only way to obtain satisfactory results is to first become intimately acquainted with the nature of the disease, whether exogenous like the southern cattle plague, or endogenous like rabies or glanders. In the first class we generally find what may be called a hæmic or blood lesion, while at the same time experience may show us that some particular organ offers more favorable conditions for obtaining the germ in a pure condition than others. For instance, I have found the liver, aside from the blood, the most satisfactory location to seek the etiological organism of the southern cattle plague; while in swine plague the spleen is the organ par excellence to be used for the same purpose. In endogenous diseases we generally find that the micro-organismal cause demonstrates its actions by certain organic lesions, or eruptions, which experience has taught us to look upon as specific, as the tubercle in tuberculosis or glanders; the parotid glands in mumps; the eruption, as it is called, in measles or scarlet. Common sense should tell us that, to use a telelogical expression, the disease has itself placed it own guide-post in these cases, telling us where we should look for the specific organism we are seeking. No one with a grain of pathological experience would adopt the course indicated in the following words from a well-known author: "Evidently an extended acquaintance with the bacterial organisms found during life and after death in the bodies of persons not suffering from yellow fever, and familiarity with the most approved methods of isolating and cultivating these organisms, would have been of great advantage to the investigator."* Were anyone to follow that advice in a primarily hæmic disease like the yellow fever, he would be a blind follower in the track of a blinder leader. In the yellow fever the blood and liver are the points in which to seek the specific organism, as in the southern cattle plague. To go to the lungs or intestines in the first search for the specific or-

* "Bacteria," Magnen—Sternberg, 1884, p. 420.

ganism in diseases of this nature would be like going to a sewer to find the germ of typhus,—we might eventually find it, but only after a deal of tedious labor. These two localities may be looked upon as sewer traps to protect the organisms,—to catch germs at the best, though in many cases they may enter the organism in one of these two ways. Having found it and become acquainted with it, then it is well enough to study its modes of entrance and dispersion and the lesions it causes at such points. I still think a most serious error has been made in just this direction in the investigations of swine plague in this country in certain quarters. In diseases of a hæmic type, it is but a natural result that severe disturbances of the circulation should occur, leading to catarrhal conditions in the respiratory tubes. In such cases a germ, or even germs, having no direct connection with the disease might still find favorable conditions to development, and be the direct cause of pneumonic complications, which have no specific connection with the disease *per se*. This occurs in the corn-stalk disease treated in the next article, where infection is positively *via* the digestive tract. The broncho-pneumonia thus resulting is simply of mechanical-irritative origin. It is impossible to call too much attention to the necessity of considering such complications at their real value. The same is true of the intestinal lesions in swine plague, which, as I have shown, while due to its germ, are really primarily dependent upon idiosyncracies in anatomical structure, and not absolutely necessary to the disease. A lesion to be unquestionably pathognomonic must never fail in being present. In this regard endogenous diseases are far more specific than exogenous, such as the southern cattle plague, swine plague, the corn-stalk disease, or the yellow fever. In hunting specific germs, there can be no better rule than that of the celebrated David Crockett, of Texas, "Be sure you are right, then go ahead." In other words, be sure of the place in which the organism sought most specifically manifests itself; seek it there, and only there. In this way, and this way only, will success eventually reward your effort.

Again, referring to the internal causes, we have diseases in which they need not be considered, not being natural or constitutional conditions peculiar to any species of animal life, though ignorance and carelessness on the part of men have well nigh made them in-bred family constitutional weaknesses, which predispose the individuals be-

longing thereto to a certain disease. This class is known as acquired internal causes. Heredity, in-breeding and carelessness should be their proper names. Tuberculosis in man and cattle—consumption—is the most striking example of this class.

After these introductory remarks we may return to the subject proper, the etiology of the southern cattle plague, and will again revert, but briefly, to its

EXTERNAL OR SUPPORTING CAUSES.

We can safely pass by those of a climatic and telluric character, because, while we know that a certain degree of temperature and a certain degree of moisture are necessary to the biological necessities of the specific cause, that is about all we do know of the climatic conditions, and we know far less with regard to the telluric, though, as I shall show, our investigations may have some indicating value in that direction. With regard to them we have discovered that certain localities only offer the necessary conditions. We know that the low and level lands offer supporting conditions to the specific cause which are not offered by the highlands in their immediate vicinity. For instance, in Georgia, the removal of lowland cattle to the mountainous regions, a distance of only fifty miles, causes eruptions of the disease among the cattle native to the same, at favorable seasons of the year.

That cattle native to many parts of our Southern States, as well as certain parts of Texas, go through an unmarked and mild form of the disease, and hence acquire an immunity from a severe attack, is also shown by the fact that cattle taken from Texas to Louisiana do not become the means of infecting the cattle native to that state.*

Cattle raised in the countries native to this disease can be removed from one district to another in the same latitude without danger of communicating the disease to the cattle that were at home there previous to their arrival.†

We know that this fact indicates that the character of the vegetation has some value in supporting the life of this specific cause, and we assume that this value is to be sought in its proneness to rapid and luxuriant growth and equally speedy decay, thus supplying not only

*U. S. Ag. Report, 1871, p. 171.
†Ibid.

nutrient elements, but protection from evaporation and shade—which is the same thing—to the germs in the ground among its roots This local-land peculiarity has been frequently observed.

"The transportation of northern cattle into Florida, Texas, parts of Mississippi, Louisiana, and South Carolina, and the traveling of southern herds across the grazing lands of the states northward, results in the sickness and death of the animals which come within the range of this singular form of contamination." John Gamgee, Report, 1871, p. 81.

That the chemical components of the earth in these southern climes, where this disease is native, have no essential value, and that the climatic conditions have, is well demonstrated by the fact that the specific germ finds equally favorable telluric conditions in the north, when the climatic conditions—temperature especially—assume the same character as those of the localities where the germs have their permanent abode. This statement will find incontrovertible support when we come to treat the outbreaks we have observed during the summer of 1887, and our own investigations.

However, the previous remarks lead to the following etiological axiom: No favorable climatic and telluric conditions, no southern cattle plague, (or yellow fever.)

It remains for the state governments where the disease permanently has its home to investigate these conditions, and not for us in the North. That much good can result, and measures adopted which will be of exceeding *prophylactic* value, is beyond question.

We have next to consider the part the southern cattle take in the extension of the disease. This discussion is naturally to be considered under two heads, viz.:

1. Texas cattle at home.
2. Texas cattle in the North.

We say "Texas cattle," because it is to them that the majority, if not all, cases of the disease in the north may be attributed.

TEXAS CATTLE AT HOME.

As previous remarks have shown, it is the opinion of many people, especially natives of Texas, that their native cattle are free from this disease. It is, however, necessary to go into that subject somewhat further.

TEXAS CATTLE ARE DISEASED.

Several veterinary authors have published statements which are calculated to give ordinary readers the idea that Texas or southern cattle are not diseased with the Texas or southern cattle plague. They do not realize that it is possible for an animal organism to become diseased in so mild a form as not to present any visible characteristic phenomena, and yet be the direct means of transmitting the disease to other susceptible animals or infecting other localities. The first is frequently the case in such a notoriously endogenous disease as scarlet, in the human family; and in the animal world, in the rinderpest among native Russian cattle, which are driven into eastern Europe and themselves give no evidence of being diseased; yet they spread that pest like wild-fire among the cattle native to the eastern parts of that country.

In the face of such well-known experiences, one would be surprised to read such an expression as the following from an authority from which we should expect quite different assertions. He says:

"Among all the communicable diseases of our domesticated animals, it would be hard to find a single one the peculiar characteristics of which make it so difficult to investigate as Texas fever."—Report of the Agricultural Department, 1883, p. 33.

To which I reply, that an easier disease to investigate than the Texas cattle disease it would be hard to find.

The same authority also says:

"The fact that apparently well animals disseminate the contagion, and that sick ones as a rule do not, is so completely at variance with what occurs in those diseases which have so far been investigated, that we have nothing in science to which we can turn with any hope of assistance." Ibid.

In the first place, as Texas or southern cattle plague is not a "contagious" disease—that is, does not originate primarily in an animal organism, or pass directly from animal to animal—even southern cattle cannot "disseminate the contagion," though they can and do act as a fitting resting place for a certain infectious principle, (which is dangerous to susceptible cattle,) and which they drop upon pastures and trails, and thus pollute them.

In hog cholera, the same thing occurs. Hogs that are thought to be well have still the means of causing the infection of other hogs by

dropping the infecting principle upon the ground, where it vegetates and becomes dispersed over the hog pens and runs.

The real peculiarity of the southern cattle plague has been, in the past, that observers have stated that its specific cause, (germ,) lost its virulent activities in passing through one generation of northern cattle, and that thereby they lost the ability of infecting their pastures, and hence could not extend the disease to a second lot of northern cattle.

This seems to be a fact substantiated by almost innumerable experiences by northern cattle owners. There is not to my knowledge a single well-authenticated case to the contrary, though there are some apparent ones to which I shall allude in another place.

With regard to Texas or southern cattle being themselves free from disease, Detmers also says:

"Native Texas cattle never contract the southern cattle fever, and possess immunity against infection so long as they remain on their native range, or north of the same, provided they are not kept long enough north to become acclimated, or in other words, have passed a winter in the North." First Annual Report of the Bureau of Animal Industry, 1884, p. 426.

But in the next passage, Dr. Detmers seems to have forgotten that he had just written, that "native Texas cattle never contract southern cattle fever," for he says:

"If Texas or southern cattle, to all appearances perfectly healthy, (which is the same as saying that they may be really, or have been diseased,) are shipped or driven north, * * * * they will infect every trail and pasture on which they graze, and every water hole out of which they drink, with the infectious principle of southern cattle fever." Ibid., p. 427.

Gamgee says:

"The conclusions, therefore, which I am disposed to draw from all the facts and arguments addressed in relation to the cause and nature of splenic fever are:

"1. That southern cattle, especially from the Gulf coast, are affected with a latent or apparent form of the disease.

"2. That they become affected in consequence of the nature of the soil, and vegetation on which they feed, and the water they drink." Ibid., p. 122.

The above does not read as if this disease was one which "has never been described as attacking southern cattle," as Gamgee had previously

asserted. But he gives still better evidence that this disease does attack southern cattle. In another place he says:

"In the South, splenic fever is distinctly indigenous, and so far as Texas is concerned, I have satisfied myself that the disease is universally prevalent in that State." Ibid., p. 119.

Again he says:

"What are the active causes in operation which tend to influence prejudicially the stamina of southern herds? Traveling over the prairies, no one can fail to be struck with the large number of dead animals to be met with. The dissection of these, or the slaughter of the first animal met with, reveals three distinct and unfavorable manifestations.

"The spleen is enlarged; the animals have, without exception, the 'ague-cake,' the stamp of a malarious district; the liver is fatty, a lesion that might be anticipated in so warm a country; true stomach reddened at its left end, its mucous membrane is eroded, or appears as if scratched with a sharp nail on its folds, and although there may be only a single and small erosion, nevertheless the traces of gastric disorder are there. I have not failed in a single instance, in Texas, and I have opened as many as twenty-six animals per day, weighing their organs carefully and watching closely for these signs. Sometimes the scars of old ulcers are more marked than the erosions on the mucous folds, and it is not uncommon to find traces of ancient lesions about the pylorus, or intestinal opening.

"From the earliest age that the calf feeds on grass to the oldest that a bullock attains, the morbid lesions alluded to may be found. They grow better and worse, and in dissecting a dozen animals one or two will be found to have blood extravasations of a very limited and delicate character in the pelvis of the kidney, in the urinary bladder, and in the intestinal mucosa." Ibid, pp. 107, 108.

As I desire to show beyond question that Texas cattle do become diseased in their own clime, although they may not present visible outward symptoms of the same, I must be excused for quoting still more from Gangee:

"That form of splenic fever which is most latent, and seen among southern cattle, is not recognizable after death by the condition of the skin, muscular system, or in many cases even by the mucous membrane, with the exception of that of the stomach. More or less, however, the blood extravasations, congestions, and blood-stained urino have been found; but these would scarcely have been noticed but for the plan suggested by me, of inspecting all slaughtered cattle and carefully weighing the spleens.

"This was done by Dr. Rauch, the then health officer of Chicago, who reports:

"'The weight, feel, and texture of the spleen, and the condition of the urine, have been found to be almost infallible in diagnosing this disease. Since the investigations commenced, over two thousand spleens have been weighed, as well as many livers.'

"The tables (see original report) give the result from the examinations of no less than 4,739 cases. These indicate that the average weight of the spleens is in excess in southern cattle over those among western steers, the excess being from half to upwards of a pound. Many Texas cattle have spleens weighing three pounds. Ibid, pp. 92, 93.

"It has been announced by stock dealers as an undeniable fact, that although Texas cattle might give the disease to natives, (northern cattle,) they were never affected by it themselves. Yet at Buffalo the commissioners found two Texas steers in a dying condition, and on being slaughtered they presented every characteristic feature of genuiue Texas cattle disease, in its most intensified form." Transactions of the N. Y. State Board of Agriculture, Vol. 2, p. 954.

" While on their way to Springfield, Ills., the commissioners sought information from all sources, and at La Fayette, Ind., spent a day in the slaughtering establishment of S. & S., where a herd of one hundred and forty-two Texas cattle were being cut up for packing.

"These animals appeared to be in perfect health, presenting no external symptoms of any disease, and the meat, except in a single instance, appeared to be of good quality. Yet the viscera of large numbers of these animals, taken indiscriminately from the herd, exhibited in every instance the unmistakable scars which that disease leaves upon the coat of the stomach." Ibid, pp. 954-5.

In one of the United States Agricultural Reports the following summing up occurs:

"There are three well-established conclusions to which these investigations have led with regard to the Texas cattle disease:

"1. That cattle from permanently-infected districts which are taken beyond this district, and where the infection does not exist, contaminate pastures, and in that way disseminate the disease among the native stock in the non-infected district.

"2. That cattle from a non-infected district, when taken into an infected district, contract the disease and suffer with the same symptoms as those which contract it in non-infected districts from exposure to the infection of southern cattle.

"3. That the native cattle of permanently-infected districts enjoy an apparent immunity from the disease, and as a rule do not present

any symptoms of being diseased on their native pasture, or when they have been driven into non-infected districts, though the most severe outbreaks may follow their importation into such districts among cattle native to the same." Report, 1885, pp. 249, 250.

The above quotation and remarks are sufficient to show that southern cattle are indeed affected with this disease, and to confirm our previously-given evidence that in its primary generation the disease is one of an absolutely local character, which peculiarity it retains when imported into a northern latitude.

SOUTHERN OR TEXAS CATTLE AS THE CAUSE OF THE SOUTHERN CATTLE PLAGUE IN THE NORTH.

That Texas or southern cattle are and have been the one cause of all the outbreaks of the southern cattle plague, either directly or indirectly, in the northern states, has been most conclusively shown in previous pages; but it becomes necessary to again revert to this question, as we wish to call attention to the importations of the cattle which were the cause of the outbreaks observed by us during the summer of 1887. Were any further evidence necessary that these animals are and have always been the cause of the disease in northern cattle, we have only to refer to the negative evidence, or the fact that not a single outbreak occurred in the Northern States during the period covered by the war of secession, when all business intercourse between the two sections of the country was positively shut off.

It is necessary, however, to call attention to the fact that in a strict sense Texas or southern cattle are not the cause of the plague occurring in northern stock. Their excreta are, however! They are simply vehicles by which inficiens is conveyed from its native heaths and transplanted on our northern lands.

SOME MORE EVIDENCE THAT IMPORTED TEXANS ARE THE CAUSE OF THE DISEASE IN THE NORTH.

In 1849 a Mr. Chilton, of southwestern Missouri, wrote:

"I have never yet known of a case that could not be traced directly to this cause.

"Wherever the native stock of the district chanced to graze upon the road-sides or pastures that had been traversed by Texas cattle, the former were in the course of two months almost entirely swept off by the disease." N. Y. State Transactions, page 1076.

MALIGNITY IN NORTHERN CATTLE.

"In just two weeks from its first appearance 24 out of 27 head were dead."

"Of 147 head, 66 have died in three weeks."

"The proportion that sickened to the whole number exposed has been two out of five." N. Y. Trans., page 1065.

"In this field were 21 head of cattle which were exposed. All died but one." Ibid., page 1067.

"Seven of eight milch cows died within three weeks of the time of exposure." Ibid., p. 1068.

"Here was a bovine pestilence that appeared to infect nearly all cattle that grazed over the trail of freshly-arrived Texans, and which destroys eighty per cent of all Northern cattle that become obviously infected." Ibid., page 1092.

"Thirty, fifty, and seventy-five per cent, and in some instances every animal, perished." U. S. Ag. Report, 1883, page 21.

THE OUTBREAKS OF THE DISEASE AMONG CATTLE AT TEKAMAH, NEBRASKA.

We must first consider the breed of these cattle, and where they came from.

Eleven hundred grade cattle (native Texans and short-horns) were purchased by Messrs. T. and L. and others, of M. & H., very extensive breeders, Fort Worth, Texas. These cattle were shipped in twenty-six cars on the 26th day of March last, and arrived in Tekamah on the 30th and 31st. One of the certificates accompanying them is from John A. Noonan, cattle inspector at Falls City, Neb., and reads thus: "That shipment has been direct from Fort Worth, Tex., except for food and water, and in cleansed and disinfected cars."

The latter point being a matter of extreme public interest, Mr. T. was asked "how this cleaning and disinfection was carried out."

His reply was:

"The cars were swept out by the railroad men at Fort Worth and then strewed with fresh sand."

One must say that is not being "cleansed and disinfected."

From Fort Worth they went to Kansas City in those "cleansed and disinfected cars," where ten car loads were really loaded into virtually cleansed and disinfected cars, because those ten cars came directly from the shops, and had never been used previously. The other sixteen "cleansed and disinfected cars" came through from Fort Worth to Tekamah.

Here the cattle were unloaded.

Were these cars then cleansed and disinfected by order of the sanitary veterinary service of Nebraska, as should have been done?

Not a thing was done with them. They were simply distributed as the railroad company wanted them. The cattle were next unloaded at the stock yards of the railroad at Tekamah, where they remained some time, and were then removed to pastures northeast of the town.

Were the yards cleansed and disinfected after the removal of the Texans?

No! and cannot be, from their very construction. While unreflecting people, and some who are prejudiced against the writer, may think that whatever strictures he may make upon our sanitary live stock service are simply in the spirit of a fault finder, he would say to them that such is not the case, but far more to cause such improvements in that service that it shall be the best in the country, and offer the most perfect guarantee possible to the great live-stock interests of the State.

Any careful stock breeder must see that these cars were never properly cleansed; and as to disinfection, there was none. It was a piece of criminal neglect to allow those cars to be distributed over the State, after Texas cattle had been in them, without first having been properly cleansed and disinfected. That no evil results followed is to be accredited more to good luck and the season of the year than to anything else.

This subject will be more fully considered when we come to speak of the proper methods of prevention.

We will now call attention to some statements which were made to the buyers of these cattle by the people in Texas in order that others may not be misled in the same manner. If these statements are true, then the result cannot be explained by any knowledge at our command.

First—The sellers positively asserted that there was not and had not been any illness among their cattle, especially the southern cattle plague, so that the buyers ran no risk whatever in purchasing them and bringing them to Nebraska. (Diligent inquiry by the buyers seemed to confirm these assertions.)

Second—That these half-breed cattle, that is, crosses between Texans and short-horns—were as liable to the southern cattle plague as northern cattle, and that they (the sellers) felt themselves in constant danger

from it, and dreaded its ravages as much as the northern people could.

There must certainly have been some gross misrepresentation somewhere!

As every outbreak of the southern cattle plague in and around Tekamah at this time followed in the wake of the Texans, and not of other cattle, of which many thousands were shipped in, the disease must have been caused by Texans and not by other cattle.

This gives rise to a poser that meets one on every side when investigating this outbreak, and that is, if the assertions of Messrs. M. and H. have any foundation, viz., that these grades are as subject to the southern cattle plague as our northern cattle, then why have they not had the disease?

The fact is, they have all been notably well, and except a few weaklings, have improved with greater rapidity than native cattle of the same age in the immediate vicinity.

If they are as susceptible to the disease as is asserted, over half of them should have died of the disease.

Again, if the cause of the disease in our cattle is really to be sought in the dejecta from these Texans which they brought from Texas, or were exposed to water or food which had been previously infected by Texans at the stopping places *en route*, then these cattle should have become ill, if M. and H. of Fort Worth told the truth.

Some additional evidence will be found later on, from the fact that a similar lot of cattle—that is, of the same grade and ages, and raised in the same locality—were shipped to Illinois two days previous to the shipment of these cattle to Nebraska, with the same fatal results.

If, as Messrs. M. and H. say, these graded Texans are as susceptible to the southern cattle plague as northern cattle, then we must assume that those sold to our Nebraska buyers were the remnants of droves that had survived attack in Texas, and hence acquired immunity to further disease, while still able to carry the disease-producing elements to our northern fields, and to infect them with their death-dealing principle.

DEATHS FROM TEXAS FEVER IN ILLINOIS.

The live stock commissioners of Illinois made the following report of the number of deaths resulting from Texas fever in that State, 1887:

"At Cartersville, August 5, 1886, thirteen died; disease contracted

from eating grass growing up through refuse from cars in which Texas cattle had been shipped.

"At Decatur, October 6th, twenty-one native cattle died; disease contracted from southern cattle shipped in for slaughter and pastured on town pasture.

"At Jerseyville, October 18th, four died; disease contracted before cattle came into possession of the present owners.

"Raymond, October 23d, five died; contracted disease from southern cattle pastured on farm.

"Effingham, July 27th, five exposed, three died, and one recovered; one failed to contract; pasture infected by manure from cars in which Texas cattle were shipped.

"New Lenuox, July 31, eleven died, two sick; contracted disease by exposure to trail over which Texas cattle had passed, and in pastures.

"Ashland, August 4th, thirty died, fifteen sick; contracted from commons and pastures where Texas cattle had grazed.

"Rushville, August 19th, four died, several sick; contracted disease from being pastured with Texas cattle.

"Effingham, August 17th, one died; contracted from litter from cars.

"Chicago, August 9th, cows from Union Stock Yards developed disease; not known where it was contracted.

"Carrollton, August 31st, twenty died; contracted from running in pasture with Arkansas cattle.

"Franklin, August 31st, four died; disease contracted elsewhere, place not known.

"West Point, September 15th, four died, seven sick; contracted from being pastured with Alabama cows.

"Winchester, September 1st, fourteen died; contracted by being pastured with Arkansas cattle.

"Quincy, September 1st, a number died; contracted the disease by being pastured with Texas cattle.

"Essex, September 6th, eighteen died, two sick; disease contracted elsewhere, place not known.

"Roseville, September 7th, fourteen died, two sick; disease contracted elsewhere.

"Nokomis, September 8th, seven died, ten sick; disease contracted elsewhere."

"There are here reported eighteen outbreaks of disease, involving a loss of more than 240 head of native cattle, besides those that died at Chicago and Quincy, where the number is not given. In twelve of these cases the loss involved would have been avoided, had the proclamation of the Governor and the rules of this Board been complied with. In the other six cases the remedy is not so apparent. The disease occurred among northern-bred cattle, as usual, and under such circumstances as to almost prove that the exposure was had and

the disease contracted before they came to this State. In such cases no inspection would avail to save the purchaser from loss, and if authorities in western states are not successful in enforcing their own laws, we shall always be subject to loss. It may be well to state, in this connection, that we have had reports from eastern states of outbreaks of Texas fever contracted from cattle passing through this State.

"If, during the coming year, we can discover any better means of protection than we now have under our laws, we will point it out in our next report.

"The cases at New Lennox present new features. These cows were placed in pasture about the middle of May. No exposure is known, except that a large drove of Texas cattle were yarded over night in the same pasture in April. These Texans were shipped from Fort Worth about March 25th, to the Union Stock Yards, and were sold and taken out March 30th. *During this time the weather was cold enough to freeze at night, and no damage was apprehended; but if they really carried the disease, it may be necessary to modify our rules relative to the time during which southern cattle may be safely moved in this State.* Of this we will report to your Excellency before the coming season. We, however, deem it our duty to add, in this connection, a few words of warning to farmers of our State who are contemplating the purchase of cattle to feed during the coming season. It will, in our opinion, be dangerous to handle cattle brought hither from those states where the southern cattle are known to be running. Disease may be contracted and no sign be manifested until from three to eight weeks have elapsed. It is then difficult to prove when the exposure occurred, or to fix the responsibility for loss. Nothing less than a full, true history of the cattle for the previous two months, will suffice to enable purchasers to buy with safety." Annual Report of the Live Stock Commissioners of Illinois, 1887, pp. 29, 30.

With regard to the shipment of Texas cattle from Fort Worth into Illinois, above alluded to, the Live Stock Commission of that State wrote me that exactly similar results followed their movements as those at Tekamah, and that the same cold, freezing weather prevailed there as at Tekamah, without having an action in checking the disease:

DEAR SIR: Yours of 8th inst. to our Mr. McChesney is at hand, also copy of "Nebraska Farmer" with your very interesting article on Texas fever, which we carefully read. We at once tried to learn the facts about shipments of Texans, as mentioned in yours, into this State. We found that a large lot were received from the region of country mentioned, but could not learn if they came from the same parties, (M. & H.) These cattle were a mixed lot, Texans and shorthorn grades. A part of them were sold and taken from the yards

March 30th to a farm twelve miles away, and there fed and kept until April 15th. During this time the weather was cold, ice being formed at night. They were driven to Kankakee county. On the first farm where they stopped over night, Texas fever appeared about July 16th. The cattle were turned into the lot used by the Texans about May 10th for pasture. Several died, and post-mortems by one of our veterinarians established the fact of Texas fever. All along their track to point of destination the same disease appeared. The owner states that none of the Texas lot have been sick, and also states that none of his other cattle have been sick. It is needless to say that we feel great interest in the work in which you are engaged, and shall be grateful to you for any facts you may be able to communicate from time to time; and if we can help you in any way shall be glad to do it.

"Very respectfully yours, JNO. M. PEARSON,
"Chm. Live Stock Com'n."

THE OUTBREAK AT ROCA, NEB., SEPTEMBER, 1887.

Fifty-eight head of graded cattle were bought by a Mr. F., of this locality, on the 28th of June, of a live-stock commission house at Kansas City, Missouri. The sellers represented them all to be native Kansas cattle, and as Mr. F. had bought of them repeatedly in previous years, he saw no reason to doubt their word. The cattle at the Kansas City stock yards are all inspected by a Missouri inspector, or supposed to be, but in this case the inspection must have been of a questionable character, as there were certainly four steers in the lot the very appearance of which would either warrant their Texas origin, or be sufficient to have justified the utmost suspicion with regard to them, and to warrant their retention and quarantine at the Kansas City stock yards. The shipment was again inspected (?) by a Nebraska inspector at Falls City in the same manner as the Fort Worth shipment into Tekamah. Further comment upon such inspection is unnecessary in this place. Suffice it to say that the southern cattle plague broke out in the course of time, as stated by the owner in the following passage:

"The cattle had had neither water nor food for two days, and when I arrived at Roca with them, on the 30th day of June, they were very thirsty. They were allowed to drink freely in Salt creek, and an hour or two afterwards twenty or twenty-five were taken sick from drinking too much water. The herd was taken on the 1st day of July to my pasture eight miles west of Roca. On the following day a steer died, and eight others died within a week. I am quite positive that all were killed by the water. The rest of the sick cattle all

recovered, and were in splendid condition up to the 29th of August. I had never had cattle do any better than they did up to that time. On the 29th of August we found two dead, and another died the following day. Then I reported the matter to the Live Stock Commission."

TRAILS AS THE CAUSE OF THE SOUTHERN CATTLE PLAGUE.

There seems to be much doubt whether cattle driven entirely by road from Texas to our Northern States have ever carried the southern cattle plague to the Northern States; but, so far as I can judge, this assertion seems to be more or less of a "say so," for the question at once arises, How could outbreaks in the north have occurred in the far Western States before the days of railroads, as they certainly did, unless from this cause? The following positive assertion is taken from the United States agricultural reports:

"In the spring of 1884 a herd of Texas cattle were driven through Cherry county, Neb., on their way to Dakota. The result was that in that county alone 2,000 head of cattle died of Texas fever during the past season. This herd traveled all the way from Texas to Dakota." Report 1884, p. 276.

The above looks like a clear case!

Other opinions upon the same subject are as follows:

"From the first it was found to be confined to the great roads or highways running through the country from the south, and finally it centered on the Texas cattle in the year 1853, by its being discovered to be confined to one highway through the country over which the cattle passed that year. On this road the disease was fatal, killing about fifty per cent of all the cattle along the road; and persons living near the water courses over which that road crossed, lost as high as ninety per cent." Report of the Board of Agriculture of Missouri, 1866.

"Many cattle have been purchased from this section of the State (San Antonio) for Colorado, and those buyers have nearly all told me that there is no danger from cattle driven over the trail from this section."

"There seems to be an almost universal belief that there would be danger from cattle which had been shipped." U. S. Ag. Report, 1885, p. 256.

I must say that my own opinion is more or less against the danger of infection from southern cattle that have been driven north from Texas, but that all depends upon the time during which they have been on the road.

THE INFLUENCE OF RUNNING STREAMS UPON THE EXTENSION OF THE SOUTHERN CATTLE PLAGUE.

Upon this subject the writer has no experience whatever, and hence must rely entirely upon the statements in the literature.

From the U. S. Agricultural Reports the following remarks have been taken:

"What makes this extension of greater significance is, that the Staunton river, (Va.,) which has held the disease in check heretofore, and continues to do so to the west of this point, has been crossed." Report 1883, p. 27.

"Charlotte county, Va. The river has here held the disease in check, and caused the border-line of the district, to turn away considerably toward the south." Ibid, p. 27.

"The newspapers of 1857–8 mention the singular circumstance that the course of the disease seemed totally arrested at the banks of any deep stream, excepting at points where Texas cattle found fording places." Report N. Y. State Agricultural Society, 1887, p. 1076.

THE CONVEYANCES OF COMMON CARRIERS AS A CAUSE OF THE SOUTHERN CATTLE PLAGUE.

Some positive evidence of this character will be found in the following letter of Mr. McChesney, of the Illinois cattle commission:

"There is no doubt, in my mind, that droppings from Texas cattle will lead to the generation of the disease in northern cattle, if spread over our pasture lands. About a year ago I was notified that cattle were rapidly dying in the town of Naples, a small place on the Wabash R. R., in the southwestern part of this State. On my arrival I found twenty-seven town cows had died within a few days, and quite a number sick, two of which died while I was there. I was satisfied as to the nature of the disease, but could find nothing to account for the outbreak until I accidentally stumbled on a bridge where the above-mentioned railway crosses the Illinois river. From a conversation I had with the bridge tender, I found that very often cattle and hogs were shipped on the same train, and that water from the tank is turned on the hogs as the train moves slowly along, and, as a natural consequence, runs into the cattle cars. On the Illinois side of the river there is about one-third of a mile of trestle work leading from the bridge, and I found, on examination, that manure from both the hogs and cattle had been washed from the cars and had fallen through the trestle work onto the grass underneath. I found, on making further inquiries, that the Naples cows were herded

in two herds, one going north of the town and the other south. The herd which went south is the one in which the disease existed, and I found they went under the trestle work to get to the river to drink. I made a careful examination of the other herd, but could find no sign of disease, neither did there any disease appear among them afterward.

"The other case occurred at Carbondale, Jackson county, this State. Carbondale is a coal-mining town, and cars in which cattle have been shipped from St. Louis are sent there to be loaded with coal. Before loading, the cars are emptied of the manure which has accumulated in them from the cattle that had been shipped in them, there being at the time of my visit an accumulation of several hundred tons, to which the cattle of the neighborhood had free access. I cannot remember the exact number of deaths that occurred, but it was in the neighborhood of forty. It was clearly proved that only those cattle which had access to the dump contracted the disease."

Another authority says:

During the summer of 1884, seven head of cattle died of what proved to be Texas fever, at Rockville, Mo. Investigation showed that no Texas stock had ever been in the town, except as they passed through on the cars *en route* for market. Further investigation showed that all the cattle that died grazed along the railroad track, where were found quantities of litter and manure, which had evidently fallen from the cars." U. S. Ag. Report 1884, p. 276.

"There can hardly be any doubt that the disease is communicated to cattle transported in cars previously used for the conveyance of Texas stock, or that cattle confined in the same pen with Texas cattle, or in pens where the latter have been confined since sharp frosts have fallen, are thereby given this disease.

"This being the case, it is demanded by the cattle men of the non-infected regions that if Texas beeves are allowed to go north during the summer months, the railroad companies be compelled to provide separate cars for the transportation of Texas stock.

"The only evidence that cars may become infected is that every spot in this vicinity where Texas cattle have been, has teemed with death to our native cattle. Permit me to use the common expression: 'I would not give a postage-stamp for a car load of cattle from here to your city (N. Y.) if a single Texas animal had been in the car for only one hour at any time from the first day of May to the first of November.'"

"Can any reason be given why cars may not become infected?" N. Y. Trans., p. 1065.

We have come now to the consideration of the most interesting question in connection with this disease, and that is:

THE SPECIFIC CAUSE—THE "CAUSA SUFFICIENS"—OF THE SOUTHERN CATTLE PLAGUE.

In order to place the question fairly before the readers of this report, it is necessary to again call attention to the essential historical data of the outbreaks at Tekamah, Nebraska.

It will be remembered that eleven hundred grade cattle were purchased by Messrs. T. and L., of Tekamah, of Messrs. M. & H., of Fort Worth, Texas, from whence they were shipped directly to Tekamah, Neb., and arrived there on the 30th and 31st of March, 1887. Also that the first indication of disease among the native cattle in and around Tekamah was in the town cows, some twenty-one of which were put in the pasture where the Texas cattle had been, after the grass had well grown—that is, after May 1st. These cattle began to die in the early days of July, and only one remains of the lot. It may be well to again call attention to the fact that not a Texas steer had been sick or died of the disease, which shows that they must, every one of them, have gone through it in the mild form common to the cattle of Texas, and that the sellers' assertions at Fort Worth were unequivocally false with regard to these cattle, whatever fears they (the cattle men at and about Fort Worth) may otherwise have for graded stock in that part of Texas, with reference to the southern cattle plague.

On Saturday, September 24th, 1887, I received a letter requesting my presence at Tekamah "at once." I immediately went, arriving at about seven in the evening, and found the population in an even greater degree of excitement than at the time of the previous outbreak in July. I was told that there were quite a number of animals sick, and that some had died, and that they were very suspicious that the disease must be extending through the influence of the sick natives that died in the early part of July last. On inquiry I also found that the sick cattle were all on three pastures, two of which place the question of the ability of sick natives to infect our northern pastures beyond all dispute, while the third is not an actually clear case.

The first of these pastures belonged to Messrs. G. and S., and was situated about six miles northwest of Tekamah.

The reader of my report upon the first outbreak of this disease, or those who still have it in their possession, may remember, or read, that it was there stated that on Sunday, June 19, 1887, twenty-four

native steers belonging to Messrs. G. and S. escaped from a pasture some six miles from town, and after wandering around were finally placed in a pasture (where some 600 of the original Texans had been confined from April 1st to the 15th), where they remained overnight, when they were again returned to the pasture that they came from, in which there were 114 other native cattle, making 138 in all. The first idea that Messrs. G. and S. had that any of their cattle were infected was in finding several sick; twenty-three of the original twenty-four became ill, two of which recovered. They found the first sick cattle in this herd on July 9th. (Fourteen died in one day.) In this case, then, the period of exposure and disease was somewhat short of twenty days, that of actual infection we cannot decide.

THE PERIOD OF INCUBATION IN THE SOUTHERN CATTLE PLAGUE.

There seems to have been, and perhaps we should be justified in saying, still is, a strange misconception in the minds of many of those persons who have been engaged in the investigation of this disease with regard to the duration of the period of incubation. They have nearly all mistaken the period of exposure, or that time which elapses from the date northern cattle have been placed upon lands infected by Texans to the first appearance of disease in them, for that of *actual infection or incubation*—that is, the period which elapses from the time the *inficiens, causa sufficiens*, enters an animal organism to the first appearance of constitutional disturbances.

This essential point of differentiation in the pathology of the southern cattle plague seems to have escaped all other observers, with the exception of those composing the Metropolitan Board of Health of New York city, upon which they say:

"Facts showing the probable period of incubation.—These facts apppear to consist of two classes:

"First. The deduction from records from the time of first exposure to the fresh trail of the infecting Texas cattle; or, the time which elapses from the first arrival and presence of Texas cattle and their excrements, to the date of the first outbreak of the disease in the native (northern) herd.

"Second. The time that may elapse between the first exposure and the first symptoms in northern cattle, when that exposure is known to have taken place, after the infected grazing grounds, or cattle trails, had become actually capable of communicating the disease. (A well

taken point, which will find its complete elucidation in future remarks. —B.) In other words, there is a distinction to be made between exposure to infection itself and the mere exposure to the Texas cattle, or to their trail immediately after they had passed." N. Y. State Trans., 1867, Vol. 2, p. 1078.

On another page of the same report the following question is asked and directly answered from practical experience. The answer, it will be seen, corresponds very nearly to our own experiences.

"What was the period of incubation in pastures that have become infected by the cause of this disease in its full destructive force?

"In about three weeks the native (northern) cattle began to die." Ibid., p. 1083.

The following remark also occurs in another place:

"When it is considered that the latent or incubative period of the disease is protracted through several weeks, always more than fourteen days" * * N. Y. Trans., p. 1101.

John Gamgee says:

"The period of incubation is generally five or six weeks." Report, 1871, p. 85.

"Thus we see that thirty to forty days usually elapse between the placing of Texas stock on a pasture and the manifestation of the disease in northern stock." Ibid., p. 88.

It will be easily seen that Mr. Gamgee included the period of exposure in the above estimate, as well as that his observations must have been upon outbreaks occurring under the most favorable climatic conditions, a fact which will find its elucidation further on.

As examples of still greater uncertainty upon this point the following are appended:

"The disease was spread by apparently healthy cattle, and these cattle infected pastures for weeks and months (! !) after leaving their native country." Report, 1883, p. 29.

A gentleman from Wyoming writes, on the period in which Texas or southern cattle may be considered safe, that —

"Experience on the border shows, beyond any question, that there is no definite time between exposure to and the development of the disease. Ninety days is as short a time as is absolutely safe." Report of Ag. Dept., 1885, p. 270.

That the last two assertions are sheer nonsense will be apparent to those who follow out this report.

There is a vast difference between the limits of the period of exposure and those of infection in any disease of this character, and that is, that the latter will always exhibit a far greater degree of constancy than the former.

In the southern cattle plague the *period of incubation is certainly under fifteen days,* while that of exposure is very uncertain. Five or six weeks, as most authorities give the period of incubation, is certainly erroneous.

In regard to the period of infection, we have a very interesting and illustrative case which happened at Tekamah.

Sept. 1, 1887, three town cows accidentally got into the pasture which was originally infected by the Texans previous to April 15, 1887. On the 21st of Sept., or after 20 days, one of these cows became very ill, but finally recovered. Symptoms the same as all the cattle that died had shown. The other two were not ill. In this connection let us again recur to the pasture where the twenty-one native cattle died. No further deaths occurred in the balance of the herd, nor were any of them sick until Sept. 21, when the owners found several dead in the same field.

This was just fifty-two days from the time the twenty-odd natives were seen to be sick after their return to this pasture from the one where they were infected; the same where the three cows above spoken of were accidentally found and infected by the Texans previous to April 15th.

Not a Texas steer had ever been upon the pasture where the second outbreak in natives occurred.

The Texas cattle could not have possibly been the cause of the second outbreak of the southern cattle plague in natives at Tekamah.

The generally advocated opinion, that the disease dies out—that is, that the inficiens loses its virulence in passing through one generation of northern cattle—is unequivocally false. Scientific and conclusive proof of the correctness of these assertions will appear further on.

To continue with the history of the second outbreak at Tekamah.

In the neighboring pastures, but on the other side of the road, were two lots of Texans. They had been there since May 15.

In one field there were nearly 600 of these Texans and one native, which had been with them since May 15, and been well during the whole time. In another field were 200 Texas steers, and with them

had been about sixty yearling natives from May 15th to the 10th of August, when half (thirty) of the yearlings were removed, to which fact we shall soon again revert. Now these natives all remained well up to August 10, and those (thirty) that were left with the Texans still remain well. Again, with other lots of this same shipmeut of Texans there have been natives ever since the middle of May last without a single case of disease occurring. This goes to show that since May 15 the Texas cattle have been given every opportunity to cause the infection of native cattle, and that they have been absolutely harmless. In fact, question as closely as you may, it is utterly impossible to find a single case of infection among the native cattle at or about Tekamah that occurred from any pasture or trail upon which the Texas cattle were, or over which they went subsequent to April 15, that is, fifteen days after their arrival. Every place where they were before that date has caused the infection of natives. This is especially true of the two pastures into which the Texans were put from April 1st to the 15th.

These facts preclnde any possibility of any Texas cattle having got into S. and G.'s herd and caused the second infection of that herd. Again, the period which elapsed between the death of the first lot of diseased natives and the first appearance of the disease in the balance of the herd was fifty-three days, which nearly corresponds to that in the first outbreak, as no town cows were put on the pastures where the Texans had been until after May 1, and in this case the time which elapsed between the first exposure and the day of the eruption of the disease should necessarily be somewhat longer for climatic reasons.

To return to the S. and G. cattle. These gentlemen having become thoroughly frightened and feeling they would lose even more heavily if they left the herd in the infected pasture, resolved to move them, each taking their own cattle to their home grazing grounds. Mr. G. left his sick ones in the pasture, while S. drove all his to his farm. This was on Saturday, September 24, the day of my arrival at Tekamah. To keep the chronological order of events we will leave Messrs. S. and G. driving their cattle home and take up my own investigations again. On Sunday morning, September 25, I drove out to the pasture where S. and G.'s cattle had been. On the way we heard that one of S.'s steers had died on the road towards town and we found where it had been buried. Upon arrival at the pasture we

at once saw one dead steer lying near the fence bordering on the road and found two others in the pasture, one of which had not been dead over twelve hours; the others were decomposed and swollen. This one we opened and we saw enough to confirm the nature of the disease. In the pasture was a brown steer in process of recovery, and Mr. G. afterwards informed me that there were some other sick ones in the same pasture which I did not see.

Monday afternoon, Sept. 26, I took a team and drove to the homes of both Messrs. S. and G. Came to G.'s place first. It was at least ten miles shorter drive than to S.'s, and what is more his cattle had to pass over an easy road. On nearing home he had one sick steer which fell down and died. He said that the balance of his cattle were still apparently well, and that they inspected them every two hours and looked them carefully over. As the afternoon was getting short and we had ten miles further to go, over a marshy road, we could not stop to inspect these cattle in person, as we wanted to proceed to S.'s, where we heard there were numerous cases. On the way we met Mr. S., who told us "to go right on" and he would return as soon as he could. So we went "on" and finally arrived at his pasture. Here we found a saddening scene indeed. The herd was made up of a grand lot of steers, mostly two years old. There were three dead ones lying on the ground, three more that could scarcely get up, and thirteen more that could easily be seen to be quite sick.

OTHER EVIDENCE THAT THE NATIVES CAUSED THE SECOND OUTBREAK OF SOUTHERN CATTLE PLAGUE AT TEKAMAH, NEB.

A clearer case of infection of the pastures by natives and the resulting infection of other natives could not be desired than the above, but we are not without other equally positive evidence of the same character.

During the July outbreak Mr. T. suffered much more severe losses than any of his neighbors from the infection of his pasture by his own Texans. His loss was mainly among a herd of eighty cows. Thinking he could put a stop to the disease by removing the cows not yet sick, in the early days of the outbreak he took the fifty remaining from the Texas infected pastures and placed them on one secure from all danger from Texans and distant from the place where the outbreak was and watched them very carefully. Nearly all

died, and were drawn off out of this pasture and buried in a deep gulch at a distance, where they were well covered. By the end of July this outbreak terminated and the pasture contained but a few recovered cattle and some horses, in which condition it remained until August 10, 1887.

Allusion has already been made to the fact that Mr. T. had sixty yearling heifers among 200 Texans from May 15 to that date and that thirty of them still remain with the Texans. Believing in the generally accepted statement that sick natives could not cause any further extension of the disease, and that pastures where they had been, though sick, or having died on them, were harmless as regards other natives if placed on those pastures, Mr. T. took thirty of these yearlings from among the Texans, most of them being thoroughbred and grade Angus, and on August 10th put them in the pasture where the natives had been sick and died and where no Texan had ever been.

On September 1st, or twenty days after the first day of exposure, several of the finest of the Polled Angus heifers were found sick, and quite a number have since died. As none were ill enough to warrant any more sacrifice on the part of Mr. T., and as we had all the evidence and material necessary we did not suggest an autopsy, as the clinical character of the disease dispelled any doubts as to its nature.

Here again we have an elapse of about fifty days from the time the pasture was first infected to the time of death among young native cattle put upon it.

The other pasture being near the town and there being a possible opportunity that some of the cattle in it had been in contact with places originally infected by the Texans, we will not consider it, though there has been quite a loss of cattle there.

These experiences having shown that about fifty days must necessarily elapse between the date of exposure of northern cattle to places infected by southern cattle, to the time of the first death, which naturally includes the period of incubation, it becomes interesting to collect statistics upon this point, which give the following confirmatory result:

OUTBREAKS OF TEXAS FEVER IN THE NORTH DUE TO TEXANS.

	PLACE AND DATE OF ARRIVAL OF TEXANS.	DATE OF OUTBREAK.	NO. OF DAYS BETWEEN.
1.	Warren Co., Ind., June 12, 1868.....	Aug. 4, 1868	52
2.	Warren Co., Ind., June 12, 1868.....	July 29, 1868	46
3.	Odell, Ill., June 25, 1868.....	Aug. 10, 1868	47
4.	Northfield, O., July 4, 1868.....	Sept. 1, 1868	57
5.	Farina, Ill., May 10, 1868.....	July 15, 1868	65
6.	Tolona, Ill., June 25, 1868	July 28, 1868	33
7.	Sodorus, Ill., June 1, 1868.....	July 28, 1868	56
8.	Champaign, Ill., June 15, 1868.....	Aug. 3, 1868	49
9.	Tekamah, Neb., April 1, 1887.....	July 1, 1887	90
10.	Roca, Neb., June 29, 1887.....	Sept. 1, 1887	63
			558

Average period between date of arrival and first notice of disease in northern stock, $55\frac{80}{100}$ days.

Dr. W. A. Thomas, an unusually exact and accomplished veterinarian, residing at Lincoln, Neb., and formerly from Iowa, informs us that Dr. Stalker, state veterinarian of that state, reports about the same general period as the result of his observations upon outbreaks of the disease in that state. These figures are to be taken as very nearly correct. In most of the cases the period must be considered as a few days too long, as it is probable, as was the case both at Tekamah and Roca, Nebraska, that the first notice the owners got of their cattle being ill was that one or more were reported to be or found dead. This may not seem probable to cattle owners in the East and western Europe, who own them in small lots and have them near home, but it is very easy to see how it can occur in the West, where grazing stock is kept in large herds, most frequently at a distance from the home farm, and scarcely ever seen oftener than once a week and then on Sundays, which seems to be the general day for salting and looking over such stock by our western farmers.

I have also a verbal communication with regard to an outbreak in native stock at LeMars, Ia. The Texans arrived on the 5th of June, 1884, and just fifty-two days afterward the native cattle began to sicken and die. It may appear singular to the reader that the previously tabulated ten outbreaks should be all that I could find in the literature upon the subject in which the date of the arrival of the

Texans in the North is exactly given, as well as that upon which the disease broke out in northern cattle, considering the number of such cases that have occurred and the immense losses that northern stockmen have suffered therefrom. Of course I may have overlooked a few outbreaks, but still I have been quite careful in looking over the accounts. One fact is made very apparent by the study of this question for any statistical evidence of value, and that is the great necessity of more extended and exact investigations of all outbreaks of this and similar diseases in this country among our stock by the live-stock and hygienic commissions of the country, as well as that the state should take some means to require every person practicing as a veterinarian or physician to gain such statistical evidence as they can, and report the same to the proper authorities.

Another thing the above table teaches, and which is of especial importance in considering the outbreaks of southern cattle plague in northern cattle, is the time at which these Texas cattle arrived at Tekamah, Neb., for unless we estimate this point at its proper value it will be utterly impossible to appreciate the importance of the second outbreak, which, as I have shown, and shall soon still more completely demonstrate, was due to the native cattle infected from the pastures originally infected by the Texans.

First. I wish to call attention to the fact that this shipment of Texans into Tekamah, Neb., occurred over a month earlier (forty-one days) in the season than any other of which we have any record.

Second. That this fact properly explains another, viz.: How it was possible for the natives infected from the pastures infected by these Texans to infect their own pastures and thus lead to a second outbreak in natives in the same pastures.

As has been said, this is the first authentic case of the kind, though there are two others quoted to which I shall refer later on; but the evidence with regard to them is insufficient to justify the conclusion that natives caused the infection of natives in them.

From these ten cases of southern cattle plague, caused in northern stock by Texans, it will be seen, by reference to the table, that the average time which elapsed between the date of the arrival of the Texans and the infection of native stock was $55\frac{80}{100}$ days; the shortest period being thirty-three days and the longest 90, that of the outbreak at Tekamah.

Before considering this point further, it is now necessary to call attention to the second outbreak at Tekamah, in which the infected native cattle were the exciting cause.

In this case we will limit our remarks to S. and G.'s cattle, as the dates of exposure and infection are more exactly fixed than in the case of the second outbreak in Mr. T.'s. It will be remembered that the twenty-four cattle of S. and G. escaped from their pasture on June 19th, and were placed in a pasture badly infected by the Texans for about twenty-four hours, when they returned to their companions in the pasture from which they escaped.

Second. That the first sick cattle were observed among them on July 9th, and that twenty-three were sick and twenty-one died. The day of the last death has not been given me, but from June 19th to July 9th (twenty days) these twenty-three cattle were infected; and, as the case proved, were capable of transmitting infection to the lands upon which they grazed.

The second outbreak in this pasture among the balance of the natives occurred about September 20th, as near as we can fix it, as the first S. and G. knew of their cattle being again infected was on the 22d of September, when they found some dead. The period of exposure and the period of actual infection are two entirely different things, both of which are subject to many influences which can exert a protracting or shortening influence.

In the Tekamah outbreak we have two cases which go to show that in midsummer the time elapsing between exposure and actual sickness is about twenty days, viz.: In S. and G.'s first outbreak it was twenty days; in the case of three town cows that got into the same Texas infected pasture September 1st, where S. and G.'s first lot first became infected, it was just twenty-one days when the first cow was observed to shorten up in her milk and lose her appetite. She recovered. The others were not sick. They were in the pasture but six hours.

The date at which the first lot of S. and G.'s cattle were observed to be sick was June 19th, and we may be justified in assuming that none of the other natives were exposed to their influences until some time after their death, viz., July 9th. The second outbreak occurred about September 20th. Allowing twenty days as the time necessary for cattle to become visibly ill, though S. and G. found dead ones in that

time, we may fix the period of the second infection of natives at about September 1, 1887. From July 9th to September 1st gives a period of exposure of fifty-three days, which is just two days and a little over less than the average in the ten outbreaks quoted above where the Texans were the primary cause. This, then, would give us 106 days as the shortest period in which Texans can infect northern pastures and the infected northerners again infect other pastures, thus leading to a second outbreak in northern cattle in the same season. As this cycle of events is so greatly open to the influences of temperature, moisture, nature of the soil, etc., it can be seen that it can be either shortened or lengthened somewhat, but never very much, or rendered impossible altogether, which has been the case in all previous importations of southern cattle into the northern states.

Temperature and the degree of moisture, that is, the lay of the land and the rainfall, are the two chief features which play the essential role in moderating the period elapsing between exposure and infection in this disease. In order to make this plain, it is necessary now to call attention to two facts:

First—The date of arrival of the Texans at Tekamah and the date of the first outbreak in native stock, April 1st to July 1st, or ninety days. This does not represent the period of actual exposure in this case, however, as the native cattle were not placed on the pastures where the Texans had been until on or after May 1, 1887, and though exposed to the infecting influences with which these pastures were contaminated, as the period between infection and actual disease must be about twenty days, it is safe to say that these pastures were not actually dangerous much before June 10th. Hence, in this case again, the period which elapsed between the arrival of the Texans and the first condition of danger in the pastures infected by them was but seventy-one days, at the longest estimate, instead of ninety days, as is apparently the case. Why this difference between a general average of 55.80 days in ten cases and 53 days in the cases of the outbreak due to natives at Tekamah, and the actual time which elapsed from the date of the arrival of the Texans to the first case of sickness at Tekamah—ninety days? These variations are to be sought in two sets of circumstances or conditions:

1. Climatic and telluric conditions.
2. The material upon which the infecting principle is bound.

With regard to the first. The Texans were placed on the dangerous pastures at Tekamah on April 1, 1887. The weather was cold and the land wet, and it continued so until about June; that is, it did not approach the conditions under which the germs find their natural development in their native fields (Texas) until about that time. Hence, until about June 1st, the germs deposited by the Texans on these fields must have lain comparatively dormant so far as these fields were concerned; or, in other words, no general infection of the fields on which the Texans were placed could have possibly occurred before June 1st.

Above it has been said that these were the first cases on record in which the disease had been extended by natives that were primarily infected upon pastures infected by Texans to other native or northern cattle.

That the contrary is and has been the generally received opinion, both by stockmen and veterinarians, can be seen by the following quotations:

"It seems that native cattle do not communicate the disease to each other, as in many instances cows were housed in the same stable with sick cows without being infected." N. Y. Trans., p. 1060. (Which is natural as the disease is not contagious.—B.)

"With but few exceptions, all those cows became infected and died that grazed near the cattle yards, or in localities formerly occupied by Texans, drinking the same water used by the latter, etc." N. Y. Trans., p. 1060.

"In conclusion I would say, that I have no reason to believe that native cattle, even under circumstances the most favorable to infection, will infect other native cattle." N. Y. State Trans., p. 1066.

"In 1868, three of a herd of sixty-five were infected and died without the disease extending to the rest of the animals."

Mr. Gamgee says, under the heading:

NON-TRANSMISSION OF THE DISEASE BY NORTHERN OR WESTERN CATTLE.

"During the three months last summer, many well marked cases have been seen of the communication of splenic fever to Illinois and Indiana cattle. At first these animals were allowed to die, but as soon as large herds of grazing stock were attacked, an effort was made to save them by shipping and sending to eastern markets. Cattle-trucks have thus been filled, in large numbers, with infected steers, which died or were slaughtered and submitted to the rendering tanks.

But not a single case has transpired to show that these animals have induced, directly or indirectly, any disease in the stock of eastern states.

"How different from this is the working of a contagious disease." U. S. Ag. Report, 1871, p. 115.

We should say so!

It has previously been remarked that Mr. Gamgee was all wrong in his pathology of this disease.

We have given abundant evidence to show that the southern cattle plague is an exogenous and not an endogenous disease. It is nothing surprising that cattle shipped directly to our eastern cities for immediate slaughter should not have been the means of extending the disease among eastern cattle, when we consider the exact climatic conditions which must prevail for a given period in order that this result may occur, and especially appreciate the fact that cattle shipped in that way and for that purpose could not possibly have had opportunity to come in contact with our eastern pastures, and hence infection of eastern cattle was rendered well-nigh impossible. The case is far different with southern cattle shipped direct from the permanent home in the south to the east for feeding and fattening purposes. In such cases the disease has frequently broken out in eastern cattle.

Gamgee reiterates his conclusion, as follows:

"None but southern cattle communicate the disease, and they rarely, if ever, do any mischief through stock yards and cattle cars, and only by feeding on pastures over which other stock roams and feeds." Ibid., p. 116.

Similar conclusions can be easily found in the other reports of the U. S. Agricultural Department, but the above are sufficient to show that the generally received opinion is and has been, that infected northern cattle are harmless as regards their own kind.

The experience in Nebraska, during the summer of 1887, that native cattle could, under exact and favorable conditions, so infect their pastures that other natives would become infected and die from the southern cattle plague, being contrary to former observations and experience, naturally called forth some criticism, but unfortunately from persons who were incompetent to give such question the scientific observation it demanded. The following remarks are a specimen of these objections:

"Dr. G.'s object was for the purpose of investigating Dr. Billings' cases of Texas fever, which had broken out among the native cattle of Tekamah, and which the latter claims was transferred from the suffering cattle in the native herd to other cattle in the same herd. This theory is exactly opposite to that of Dr. G., who has always claimed that it is impossible for the disease to spread among the cattle of native herds to other cattle in the same herds."

"Dr. G. said: Dr. Billings has done more to injure the cattle business of Tekamah than any other one thing. He has spread his opinion in regard to the contagion of the Texas fever broadcast, and in consequence, buyers or purchasers cannot be found in that part of the country. I have always been of the opinion, and am of the same opinion yet, that Texas fever will not spread in a herd of native cattle. The way the malady strikes our native cattle is from the southern cattle. When the latter come north they do not die from the effects of it, but the native cattle are almost sure to die from it. As far as my observation and experience reach, the malady cannot be transmitted from native cattle to other native cattle, and owing to the theory advanced by Dr. Billings, the people of Tekamah are alarmed, and the cattle business there is almost stagnated."

"There are several other things that also might be taken into consideration. The native cattle are known to have come over the same trail that the Texas cattle were on, and they may easily have caught the malady there. Then, again, the high winds may have blown the dry manure from the pasture of the Texas cattle to that of the native cattle, and the disease could have been generated in that manner."

This observer says that native cattle cannot extend the disease.

In the first place, he seems utterly unaware of the fact that were this the case, the southern cattle plague would be a *unicum* in all our pathological records of diseases of an exactly similar character. Persons who have never had any scientific education are totally unfitted to value experiences of this kind, and, as is to be easily seen, fail utterly in appreciating the relation between climatic influences and the development of this plague.

There are but three authorities upon this subject who have expressed any opinion whatever.

They are:

1. The Metropolitan Board of Health of New York city, who studied the disease in a very exhaustive manner. Their report is to be found in the transactions of the New York State Agricultural Society, 1867, vol. 2.

What do they say?

"That the evidence of the contagion of the Texas cattle disease being communicated from native to other native cattle in this group is beyond a doubt." Page 1047.

While the above quotation certainly speaks against the assertions of the late state veterinarian of Nebraska, and very positively contradicts the generally-received opinion, still I cannot accept it as testimony in favor of any of my own observations.

By referring to page 1043 of the N. Y. State Transactions, we shall find the history of the cases upon which this conclusion was based. It is as follows:

"On the twenty-fifth of August there arrived at Hamptonburg, Orange county, N. Y., forty-four head of native cows and heifers, directly from Painesville, Lake county, Ohio."

On page 1046 we read:

"Cattle, that were supposed (! ! ?—B.) to be natives of the western states, and that had arrived from Painseville, Ohio, * * * began to show symptoms of disease within a week from the time of their shipment from Painesville, O. The true Texas cattle disease * * * was prevailing at the time in Summit county, Ohio. That these cattle, which were at least reputed to be native stock, (! ! ?—B.) did communicate the same kind of fatal disease to native stock, that killed several of their number, does not admit of a shadow of doubt. In the absence of those exact methods of investigation which the medical anatomists pursued in our cities, we do not hesitate to take the straightforward testimony of experienced herdsmen, who saw the disease and described it to us."

Upon such testimony is based the assertion, "That the evidence of the contagion of the Texas cattle disease being communicated from native to other native cattle is beyond a doubt."

I will at once place this question "beyond a doubt," that the disease occurring in the native Orange county cattle was not the southern cattle plague, and that the "testimony of experienced herdsmen" was valueless in this, as it generally is in all such cases.

In our consideration of the period of incubation in this disease it will be remembered that we quoted the following from the same report:

"When it is considered that the latent incubative period of this disease is always more than fourteen days."

It will also be remembered that we consider it to be always under

15 days, and that we have given evidence showing the correctness of this conclusion; for, if in cases where infection has resulted immediately on exposure, the first death occurred in twenty or twenty-one days in several such instances, we must make allowance for the time which elapsed for the generalization of the inficiens and its action, which under natural infection I think takes not less than five days, then the period of infection (visible) must be about fifteen days (or less) to death.

Again, it has been shown conclusively, by statistics and my own observation, that the average time elapsing between exposure and the first death has been 50 odd days, the shortest period recorded being 33 days.

Now let us compare these facts with the results observed in Orange county, N. Y.

The Ohio cattle arrived Aug. 25th. "Aug. 29th two cows were purchased from the same lot, by Mr. Wm. Moul—apparently well." Sept. 8, one of these cows died. "On the 12th of Sept., 13 days afterward, one of Mr. Moul's milch cows was found to be ailing." On the 14th (15 days afterward) another cow was sick; on the 19th still another. "These cows were unfortunately not examined after death." "The cows that died, of the original herd, were not buried sufficiently deep, their carcasses becoming exposed. Two pairs of oxen and two young heifers were allowed to pasture in this field where the dead cows were buried. About two weeks after one of the oxen was found sick, but finally recovered. Another ox was found dead on the 21st of Sept. Oct. 24th, two heifers, which had been pasturing in the same field where the dead cows lay, were found dead. They had not been discovered to be suffering with any disease." Ibid., pp. 1043, 44, 45.

The above does not conform to the southern cattle plague, but does to anthrax, which disease has frequently been confounded with the former.

The next case which apparently goes to show that natives can be the cause of the extension of the disease to other native cattle is from Dr. John H. Rauch, once health officer of Chicago, and at present the noted and efficient secretary of the Illinois board of health, a man of no mean authority on this question. He says:

"With regard to the transmittal of this disease by native to other native cattle, I must confess that notwithstanding the weight of testimony against, I am inclined to believe that it can, and does take place.

"General instances of this character fell under my observation, but the most conclusive evidence I have is that native cattle were purchased in Chicago in August, and taken to Lebanon county, Pa., and that a short time afterward they died, and that other native cattle on the same farm and neighborhood died, and that no Texas cattle had been near the place." New York State Transactions, p. 1082.

The evidence is also not conclusive in this case, for the same reasons given above.

Mr. Salmon, chief of the bureau of animal industry, has also something to say on the subject, which is as follows:

"Are pastures ever infected by sick natives?

"If the observers of Texas fever are practically unanimous in concluding that the disease is never conveyed from one animal to another, this is far from being the case in regard to the ability of sick animals to infect pastures. It is true that in all the observations of 1868 there were but two cases (those quoted) where it seemed at all certain that pastures had been poisoned by sick northern cattle. In regard to these, however, there was little chance for doubt.

"In my own observations I have generally found that sick natives were harmless, but there seem to be occasional instances, particularly where they have been pastured on infected lands, in which they carry the poison and infect lands that were previously safe." Report 1883, p. 61.

Mr. Salmon then gives cases which he considers to be proof of this statement.

The above is valueless, and Mr. Salmon entirely knocks the bottom from under his argument when he says, "particularly where they have pastured upon permanently infected lands," which reduces them to the same condition as southern cattle, and the cases quoted by him are precisely of that character as they occurred in Virginia, in places where Mr. Salmon is showing, or endeavoring to show, that the line of permanent infection is gradually extending northward.

With regard to the assertion that "the high winds may have blown the manure from the pasture of the Texas cattle to that of the natives, and the disease could have been generated in that manner," we have the incontrovertible fact before us that with those same Texas cattle had been 60 natives from May 15th to Aug. 10th, and thirty of the lot remained until they were all removed from the pasture in October, not one of which ever became ill. Again, if a herd of such Texas cattle can be in a pasture which will kill natives if put on it, and yet sur-

rounded by natives in other pastures, how is it that the wind has never acted in that way, except as it finds its generation in the brains of just such ignorant people as the author of such a statement, in the numerous outbreaks that have previously occurred in the North?

How is it that "a fence will keep it off," as every one knows to be the case?

By what means then do southern cattle infect our northern pastures, as well as natives cause the extension of the disease when it occurs?

THE EXCRETA—THE MANURE IN THE FIRST DEGREE AND URINE IN THE SECOND—ARE THE ONLY MEANS BY WHICH THIS OCCURS.

Dr. H. J. Detmers puts forth the totally indefensible hypothesis that it is the saliva of cattle by which this infection occurs. As his remarks upon this subject are very interesting, we cannot do better than quote them in detail, though portions of them have been previously noticed, from the U. S. Agricultural Report of 1884:

"Native Texas cattle never contract the southern cattle fever, and possess immunity against infection as long as they remain on their native range, or north of the same, provided they are not kept north long enough (in any of the Northern states) to become there acclimated, or, in other words, have never passed a winter in the North. But the same cattle, if taken from their native range and driven or shipped south, will gradually lose their immunity in proportion to the distance they go further south, and thus, if going far south, finally become liable to be infected and to contract the fever. This shows the infectious principle must be the more intense the further south the locality.

"If Texas or other southern cattle, to all appearances themselves perfectly healthy, are shipped or driven North, away from their native range, after new grass has appeared and become interwoven or intermixed with the old dead grass of last year's growth, which, owing to the warmer weather and the usually abundant rains of the early southern spring, is in a decaying condition, and these cattle, thus compelled to eat both the intermingled old and new grass, have but once taken a good meal of this mixed herbage, they will as soon as they arrive at a certain latitude further north infect every trail and pasture on which they graze, and every water-hole out of which they drink, with the infectious principle of southern cattle fever. And the native northern cattle following them will, after some interval of time (period of incubation), contract the disease, as a rule, in its most fatal form.

"If Texas or other southern cattle are moved to the North before any new grass has made its appearance on their native range, or rather before the dead grass of last year's growth has commenced to decay, no infection of northern pasture, etc., will take place, no matter how far north the southern cattle may be shipped or driven. If, however, the cattle thus leaving their native range in the South early in the season, or in the winter, should travel slow enough to be yet within a part of the South in which the southern cattle fever has its permanent source, when warm weather and abundant spring rains cause a decay of the old grass and start a vigorous growth of the new, the effect will be precisely the same as if the cattle had been kept that long on their native range; only the infectious principle imparted to the northern pastures, etc., may be a trifle less virulent, and taken up by northern cattle may cause a somewhat milder, though in a majority of cases yet fatal, attack of the disease. I had repeated occasions to observe that the fever, as a rule, is the more severe the further south the source of the infectious principle.

"Northern cattle shipped to Texas, or to other parts of the South, will contract the disease, and as a rule die of it, if only once pastured soon after their arrival on land that contains both old and new grass—particularly if it is so-called hog-wallow land—or if only once allowed to drink out of a water-hole receiving the drainage of such land.

"Grown northern cattle imported into Texas usually contract the disease with more certainty, and in a more fatal form, than imported northern calves and yearlings. Whether such is the case because the latter have a smaller mouth, are more dainty eaters, and better able to pick out the blades of grass they want, and to refuse what they do not like, or whether their young organism is better adapted to resist the influence of the pathogenic principle, I will not now decide, and will only mention that some young animals, even calves, contract the disease in just as acute and severe a form as full-grown cattle.

"In the North—say north of the southern boundary line of Kansas—the disease is only communicated through trails, pastures, and grazing grounds, or rather their grasses and other food-plants, and water-holes previously infected by southern cattle; but it usually does not make its appearance until the latter part of July or in August, or until the northern prairies, fields, and pastures, owing to the heat and often abundant rains of the summer, contain a comparatively large amount of vegetable débris or decaying vegetation, which, it seems, is an important factor in propagating the pathogenic principle if once deposited. That a propagation of the once deposited pathogenic principle actually takes place on the grass or herbage of the trails, pastures, or grounds, etc., and outside of the animal organism, is demonstrated by the fact that the period of incubation, as a rule, is a long one, if the native northern cattle immediately, or within a few days,

follow the southerners on the trails, pastures, etc., while it usually is considerably shortened if a few or several weeks intervene between the time at which the southern cattle left and the time at which the northern cattle entered the infected premises. As, however, the infectious principle is not volatile, and is not disseminated through the air or by winds, its propagation on the grass and herbage of the infested grounds may not be the sole cause of shortening the period of incubation, and the difference just stated may also, to a certain extent, be accounted for by the following fact: In about two, three, or four weeks after a herd of cattle has left its grazing grounds (trail, pasture, prairie, etc., as the case may be) a fine crop of young and juicy grass will be found, if the season is not unfavorable to its growth, wherever the cattle have grazed; while at all those places or spots where they have not been grazing the grass will be comparatively old and tough. If a herd of native or northern cattle immediately follows a herd of Texas or other southern cattle, which have infected the premises with the pathogenic principle of southern cattle fever, the former will principally graze where they find grass, and not where the southern cattle have cropped it, and where they, at the same time, have deposited, as I shall explain further on, the infectious principle. But if the herd of northern cattle enters the pastures, etc., formerly occupied by the southern cattle two, three, or four weeks after the latter left them, or after a new crop of young grass has made its appearance, the former, for obvious reasons, will prefer to graze at the very places where the southern cattle have grazed, and deposited the pathogenic principle. As it is well known that the length of the period of incubation depends, to a certain extent at least, upon the quantity and intensity of the infectious principle taken up by the animal organism, no further explanation will be necessary.
* * * * *

"If all the facts known in regard to the communication of southern cattle fever to northern cattle by means of trails, grazing grounds, pastures, water-holes, etc., are duly considered as they present themselves, there can hardly remain a doubt that the infection of the trails, pastures, etc., must be effected by means of the saliva of the southern cattle.

"In proof of this assertion I may be allowed to state a few facts, and also to dwell upon other theories now and then advanced. First, as to the latter. One theory charges the infection to a deposit of the urine of southern cattle. If it were the urine that causes the infection, only those comparatively small and far apart spots in which the urine of southern cattle is deposited would be able to communicate the disease to northern cattle, for it has been established beyond a doubt that the infectious principle is not carried through the air or disseminated by the winds, and that even a wire fence separating a pasture occupied by northern cattle from a trail or pasture of Texas

cattle is ample protection. Besides, cattle are not apt to graze where another animal has urinated, and as the urine is soon absorbed into the ground (very favorable to the infection of the same—B.), or evaporated, it could never be explained how it can be possible that the infectiousness of a pasture or trail increases in intensity at least for several weeks after the southern cattle have left it. (The explanation is easy: by the multiplication of the germs in the urine, and they are there!!—B.) If the urine constituted the vehicle of infection, the wholesale infection of every northern herd of cattle that passes over and grazes on a trail of southerners, or feeds on a pasture that has been occupied by the latter, would hardly be possible, and at the utmost only one or a few animals of a herd would contract the disease. Another theory charges the excrements of southern cattle with constituting the vehicle of the pathogenic principle. The objections just made against the urine theory will also dispose of the dung theory; besides, all cattle, but particularly grown animals, carefully avoid to graze where other cattle have deposited their excrements. They are apt to sniff at places where horses have voided their dung, and when suffering from certain digestive disorders, attended with a vitiated appetite, may even eat some horse manure, but they will never graze, if they can help it, where the dung of their own kind has been deposited, a fact well known to every cattle man. It may be possible that some pathogenic bacteria pass off with the dung, or even with the urine; but if they do, they most assuredly do not furnish the principal source of infection. Another theory charges the hoofs of the southern cattle with being the communicators of the infectious principle. This theory, too, can be easily disposed of, even if it were possible that the hoofs were able to take up the pathogenic principle (bacteria, for instance) at the native range and convey it to some other place, that other place could only be in the immediate neighborhood, because at every step in the grass the hoofs are wiped, and in mud or water they are apt to lose whatever may cling to them; besides, neither the horn of the hoof nor the skin of the foot constitutes the soil or medium needed for the reproduction, preservation, and propagation of such a pathogenic principle as that which causes the southern cattle fever. Even if the skin of the foot, particularly in the cleft between the hoofs, constituted a suitable medium, and afforded all the conditions necessary to the existence and reproduction of the pathogenic principle, the constant wiping and friction which those parts are subjected to on the march would preclude the possibility of conveying the principle (bacteria) in that way a thousand miles, or even further. Still another theory, which has a great many adherents even among practical cattle men, charges the ticks often found on Texas cattle with being the bearers of the infectious principle, or even with constituting themselves the pathogenic agency. The principal objection that can be brought to bear against this theory is the fact that southern cattle free from

ticks will infect northern pastures, etc., just as soon as those that have them, and that ticks of the same kind also occur in countries in which the southern cattle fever never originates or makes its appearance, unless it is introduced by southern cattle infecting a trail, pasture, water-hole, etc. The perspiration (through the skin) of southern cattle, and even the expirations (from the lungs) have been accused of constituting the pathogenic principle, or the vehicle of the same. But this theory, too, is fallacious, for if true, the pathogenic agency would be of a volatile nature, and be communicated through the air, which it evidently is not, as already stated. Hence the only thing that remains as the probable vehicle and medium of the pathogenic principle is the saliva of southern cattle, deposited by them, not only wherever they graze and drink, but also often dropping in strings from their mouths on the march."

Before personally considering Dr. Detmer's saliva theory, let us see what have been the conclusions of other observers:

"One of the most reasonable suppositions in regard to the infection of pastures is, that this occurs from the excrement of southern cattle.

"To test this theory, cattle excrement was taken from Savannah stock yards, placed in tin cans, and used for inoculating purposes within three days." Report, 1883, p. 39.

The results were negative!

The reader will, however, soon have the most positive evidence placed before him that the manure is the chief means by which the land is infected in this disease.

On this point Gamgee says:

"It is not the breath, or saliva, or cutaneous emanations, which are charged with the poisonous principle, but the fæces and urine." Report, 1871, p. 118.

The New York report says, "That the effort to discover the precise nature and sources of the infective carrier of the pestilences which are spread by means of excrement, as in the case of cholera, typhoid fever, and this disease of cattle" (p. 1153) shows that these authorities also looked upon the manure as the chief means by which the land is infected.

See also the previously quoted letter from Mr. McChesney, of Illinois Live Stock Commission.

One is surprised to read such conclusions as the above from a person who has done such good work and come so near arriving at the truth as Detmers did in regard to hog cholera.

It is incomprehensible to me how a person of Detmers' cleverness could have made such an assertion as:

"As the urine is soon absorbed or evaporated, it could never be explained how it could be possible that the infectiousness of a trail or pasture increases in intensity, at least for several weeks after southern cattle have left it."

This conclusion of Detmers' is even more ridiculous when applied to the manure.

The "increase in infectiousness" is easily explained and absolutely in correspondence with the biological phenomena of germ life.

The "increase in infectiousness" is caused by the active proliferation of the germs in the manure and urine, which continues so long as the climatic conditions are the same as those of Texas, and gradually extends to the surrounding ground, finding especially favorable and protecting conditions in the looseness and moisture of the soil, due to effects of the roots of the herbage, and its protection of the soil by the parts above the ground. That the infecting abilities of the urine are less than those of the manure is certain:

1. Because it contains less germs.

2. Because it soon evaporates, and thus offers less favorable means for their protection, but it does offer some by adding moisture and favorable chemical components to the earth upon which it falls.

That the dung is really the vehicle by which our northern pastures and lands become infected is so palpable that it seems beyond comprehension how any one can argue against it. Detmers himself gives the strongest possible evidence in its favor, though rejecting the logical conclusion, when he says:

"If Texas or other southern cattle, to all appearances perfectly healthy, are shipped or driven north, away from their native range, after new grasses have appeared and become interwoven with the old dead grasses of last year's growth, which, owing to the warmer weather and usually abundant rains of the early southern spring, is in a decaying condition, and these cattle, thus compelled to eat both the old and new grasses, have but once taken a good meal of this mixed herbage, they will (as soon as they arrive at a certain latitude further north) infest every trail and pasture on which they graze, and every water-hole out of which they drink, with the infectious principle of southern cattle fever." Report, 1884, p. 427.

The lay reader might be led to think from the tenor of the above passage that it was the grasses directly, the "mixture" upon which

Detmers lays so much stress, that caused the infection of our northern lands. A more mistaken conclusion could not be made! The old grasses, wilted and matted together down on the southern plains, form a beautiful natural carpeting to protect the ground underneath. They draw little or no moisture from it; they protect it from the cool influences of the air and evaporation, and thus serve as a natural means to preserve the vitalities of the germs lodged in the ground. If cold enough, the germs may remain dormant, both in the roots of this old dry grass and the ground, until the "warm weather and usually abundant rains of the early southern spring" supply just the conditions these germs have been waiting for to take on renewed life and activity.

On the other hand, as these germs must undoubtedly pollute the herbage, dry and withered though it may be, the moment the cattle commence to eat it (and when do they not eat it in Texas?) and the cattle take it into their stomachs, these microphytes find there the necessary temperature to development, and suitable conditions, though not so favorable as in the earth itself, and there they develop and increase in numbers until they permeate the whole mass. The very catarrhal condition of the entire inside of the intestinal canal found in this disease serves as a means of protection to them, especially through their journey through the fourth or digesting stomach, from which they get into the intestines and are passed off and dropped on our northern lands, where they find protection in the manure, by its volume, by the heat produced in its fermentation, by the drying up and hardening of its outer surface, until they are scattered about by the feet and movements of the cattle, by the rains, by winds, and by the disintegration of the heap.

That Detmers' saliva theory has little or no foundation is shown by his own argument with regard to the grasses. The grasses do not come out by way of the mouth. Cattle do not ruminate much when being driven rapidly over a trail. Saliva does not come from the stomach, but from the glands within and around the mouth, hence their drewlings have little or no opportunity to become profusely contaminated with germs, and by dropping on the ground infect it.

The manure, on the contrary, is being dropped almost constantly by a large herd when on its passage or in a pasture.

The same argument used by Detmers against the urine as an infection-bearing material is still more conclusive against the saliva.

The saliva must drop in even smaller quantities, and hence be only found upon the surface of the ground and herbage, and thus evaporate still more rapidly; and, considering the nature of the urine in this disease, it is still more probable that it is replete with germs than the saliva.

The facts in connection with the outbreak at Tekamah are of themselves sufficient to contradict Detmers' saliva theory completely, and point to the manure only as the means by which our lands are infected by the Texans.

The Texans arrived March 31st, 1887; from that time until April 15th, at least, the weather was very cold and quite wet, in fact it froze hard quite a number of times, and there were frosts nearly every night. Now, it is an unquestionable fact that such freezing and frosts will kill the germs of the southern cattle plague if the cold comes in direct contact with them; that is, if there is not sufficient protecting material over and around them. The experience in some parts of Texas shows this: When the germs in the grasses and on the surface of the ground are killed, but not those in the earth itself, for the disease again breaks out the next summer, the frozen crust on the surface of the earth keeping the warmth below. Now, were it the saliva in which the germs were lodged and by which it is planted in our northern pastures, it must have laid on the grasses and surface of the ground only, at Tekamah, and here the germs must have been killed by the cold weather that prevailed for over two weeks, and then no outbreak of the southern cattle plague would have resulted.

With these remarks we may drop Detmers' saliva theory.

THE GERM OF THE SOUTHERN CATTLE PLAGUE.

Every important discovery has some history which belongs to it, and which shows that ideas and investigations have early centralized about a certain focus until the efforts of many have finally crystallized into the discovery of the object sought by some one person.

In this regard the history of the southern cattle plague has not varied from the general course. Work in this direction was first undertaken by the metropolitan board of health of New York City, but without any successful result, although they felt quite confident to the contrary, as the following shows:

"Bringing his skill and large experience to the work, Professor Stiles soon unmasked the mysterious and active agent in this disease, revealing not only its perfect outline and form, under the miscroscope, to every eye, but, with equal distinctness, revealing the mode of its attack upon the blood discs and the entire destruction of the blood that resulted from it. One of the most brilliant discoveries in medical science, but not more brilliant than useful." Report of the New York State Cattle Commissioners, Transact. N. Y. Board of Ag., 1867, p. 951.

"The parasite that is found in the blood and bile of infected cattle.

"Whether we regard it as a propagating and destructive cause of the disease, or simply as a concomitant, it is necessarily an important attribute of the pathological or destructive agency that operates upon the blood. The prolific brooding and growth of the fungus (micrococcus) is wholly dependent upon the living elements of the blood for its soil and food to grow upon. But the real significance and value of the results that have been reached in the researches upon this collateral element of the inquiry into the disease, promise much for practical hygiene as well as for herd farming; for such complete demonstrations will lead to a kind of absolute knowledge that is much needed concerning the pestilential epidemics, as well as the destructive epizoötics, and will lead to their entire protection." Ibid., p. 1167.

What was this germ?

Dr. Stiles, in his contribution to the above report, says:

"Quite early in this investigation my attention was attracted to the existence in the diseased bile of minute vegetable germs, which multiplied abundantly in the various specimens of bile preserved for analysis. They existed in the form of spherical or irregular aggregations of micrococci, the nature of which could be determined only by the employment of the highest powers of the microscope and by studying their development.

"They were found in fresh blood and bile, but with difficulty.

"In specimens of bile collected in the evening they would be found abundantly in the morning, the white color of their aggregation contrasting with the yellow hue of the flocculi of the bile to which they were attached and from which they seemed to be derived, their abundance being such as to preclude the idea of their derivation from any other source than the blood or bile itself." Ibid., p. 1141.

These quotations will answer for our purpose. They show that the New York investigators concluded that a micrococcus was to be looked upon as the cause of the southern cattle plague. It is not to be expected that these conclusions should be proven to be correct when we consider the development of bacteriological research at that

time, 1867, when no trustworthy methods of isolation had been perfected, nor had our present technique of differentiation by coloring for microscopic examination been applied to micro-organisms.

In the year 1871 the United States government authorized Mr. John Gamgee to study this disease, as has already been noticed many times in preparing this report. The microscopic examination of the blood and tissues was largely made by Dr. John S. Billings and Dr. Curtis, surgeons in the U. S. army. Their results were unsuccessful in demonstrating the presence of any specific germ, but are very interesting, as they mark a period in the development of this peculiar department of scientific work. It should be remarked in justice to the gentlemen named, that neither of them would to-day endorse the views they then promulgated, as they are directly contradicted by our present experiences. They say:

"In cases of splenic fever of cattle our experiments, therefore, fail to establish the presence of any peculiar or special cryptogamic germs in the blood, and instead of supporting the notion that the micrococcus granules which are present in any way cause the disease, tend rather to show that their occurrence should be considered as an effect of the malady, whether constant and inherent, or altogether fortuitous; for since these granules, if fungus in their nature, must be, as indicated by the cultivation, forms of the very commonest moulds, it is certainly a much more probable hypothesis that the disease so destroys the vitality of a part of the blood as to render it capable of supporting and nourishing a low form of ubiquitous fungi which perish when introduced into a healthy subject, than it is to imagine a deadly disease, occurring only under certain rigidly prescribed conditions, as caused by the presence in the economy of the germs of fungi notoriously harmless and of universal occurrence.

"It is, of course, possible that the fungi developed in the fluids of a diseased animal and became carriers of contagion.

"The statement of Dr. Stiles, that the fungus origin of zymotic diseases is now conceded by the highest authorities in mycological research, will no doubt surprise the said authorities, for the highest authorities in England, America, and Germany concede nothing of the kind."

From the above it will be seen that the United States authorities were very wide from the mark, and that they absolutely failed to appreciate the true direction of modern research, which was so well seen by Dr. Stiles and the medical men of the metropolitan board of health. Dr. Stiles' observation quoted above, that "the fungus origin

of zymotic diseases is now conceded by the highest authorities in mycological research," though written in 1867, would now be endorsed by the "highest authorities" in every country, and probably by no one more fully than Dr. John S. Billings, the erudite librarian of the medical library at Washington.

The fact that all sorts of fungi followed the cultivation experiments of these gentlemen, is not be laid to their door, but, as said before, to the fact that a proper technique had not been then developed, as has since been the case, thanks to the practical genius and untiring energy of Koch and other European workers.

The next work which we find upon this disease and its germ is from the hands of Mr. Salmon, of Washington, and published in the Report of the Department of Agriculture, 1883.

Of Drs. Billings and Curtis' work, he says:

"Billings and Curtis also concluded that in the blood, bile, and urine of cattle slaughtered in Texas, apparently healthy while alive, but presenting after death the appearances characteristic of the splenic fever, there are present minute bodies corresponding to the micrococcus of Hallier, which exhibit the same behavior with re-agents as the spores of fungi." p. 34.

The passage quoted has its value completely nullified by the quotation that has been previously made, where Drs. Billings and Curtis say emphatically:

"In cases of splenic fever of cattle, our experiments, therefore, fail to establish the presence of any peculiar or special cryptogamic germs in the blood, and instead of supporting the notion that the micrococcus granules which are present in any way cause the disease" * *

With regard to this search after the germ of this disease, Salmon says:

"Of course in my search for the disease germ it has been a primary object with me to determine if the blood of affected animals really contains either bacteria or spores of any kind of fungi. * * * In none of the specimens of blood which I have tested have I ever been able to discover any living germs, and if the plague (Texas fever) is due to a parasite this does not multiply in the blood before death." p. 34, Report 1883.

Wrong, as usual!

"In bile from animals affected with Texas fever the diplococus which I shall soon describe as existing in the spleen, may be seen floating about with bacilli and bacteria.

"The bile then is not a suitable liquid in which to look for pathogenic germs, but they may or may not be connected with the causation of the disease, as there is no possible method at command for deciding this point."

With regard to observations of Drs. Stiles, Billings, and Curtis upon micro-organisms in the above named fluids, Salmon says:

"If (they) found micrococci in the blood and bile then, the discovery throws no light on the pathology of the disease, for those found in the blood were either granules of débris, or atmospheric bacteria, and those in the bile were in no way differentiated from the numerous septic forms which are always present in that liquid." pp. 34, 5, Report 1883.

Salmon tells us that his materials "were always obtained with the greatest precautions, vacuum tubes being filled directly from the veins and immediately sealed." p. 34, Ibid.

In spite of all these precautions he did not succeed in getting pure collections of germs, for he says that the "*liquids contained the most diverse organisms,*" referring to the gall, while in the blood he found *nothing.*

I will briefly say here that the micro-organism which I have found in the southern cattle plague (in the outbreaks of that disease, at Tekamah and Roca, Neb.) in the summer of 1887, was always pure and alone in the blood and gall, as well as the organs of each animal which I had killed and which was subjected to examination. Also that the same organism has been invariably found in the blood, gall, and tissues of the ground squirrels which were used for the experimental test of its virulent properties.

This object is not a "diplococcus" as claimed by Salmon!

As to what he found, Salmon says:

"Micrococci of the spleen and liver.

"Two cultivation tubes were infected with some pulp from the spleen. The next day both were turbid, and something had evidently multiplied in them. One was examined and found to be a pure cultivation of diplococci, without any power of movement; they resembled the fowl cholera micrococci, but were smaller.

"Sections prepared in this way—stained with aniline violet—plainly showed granules of the dumb bell or figure eight form, which were stained a different shade from any part of thet issue." Ibid., p. 35.

In order to obviate any misunderstanding on the part of Mr. Salmon, or the reader, I wish to lay emphasis upon two statements

in the above quotations, though I shall have to refer to them again later on.

1st. *Mr. Salmon tells us that the micro-organism seen by him,* in the tissues of the liver and spleen of cattle that had perished from Texas fever, *is a "diplococcus;"* that is double coccus.

2d. That it had a *"figure 8 form;"* that is, constricted in the middle of its body.

3d. That it was "without any power of movement."

These three points are very essential to be remembered.

Opposite page 36 of the report of 1883 may be seen an illustration of the object described by Dr. Salmon, which is reproduced on the adjoining page of this report), who further says:

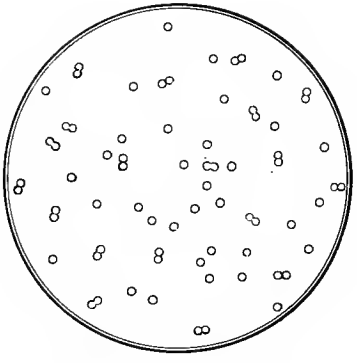
"Owing to the difficulty of obtaining suitable cases, these were the only reliable investigations of the splenic liquids, but they are sufficient to prove that a parasitic schizophyte multiplies in the spleen in cases of this disease.

"I have no desire to exaggerate the importance of this discovery. It undoubtedly needs confirmation, but this I feel assured will not be lacking, if the investigation is continued through another summer." Ibid., p. 36.

The confirmation has never been given, and so we may be justified in assuming that this cocci went the way of that of hog cholera, and was allowed to pass into a "innocuous desuetude."

Salmon made some experimental inoculations with his "diplococcus," but all he says with regard to them is that he "inoculated a cow, a small heifer, and steer on the afternoon of Oct. 3d, with material from a cow that he had killed in the last stages of the disease." This cow died, but he gives no evidence as to the complaint she died of, and as "in the young animals there was (not) any serious sickness," and as no autopsy notes are recorded, which would certainly have been the case had there been any reason to suspect the southern cattle plague, we may safely conclude that the animal did not die of that disease.

Neither does Salmon give us any evidence that his "diplococcus" had any pathogenetic action when inoculated upon rabbits, mice, and other small animals used in experimentation, and as he gives evidence that he was accustomed to use these animals in the study of hog cholera, it is also safe to conclude that his "diplococcus" was a failure, like his "micrococcus" of swine plague. He admits all this, by saying of his experiments that "they are certainly negative," though



Salmon's Germ of Texas Fever.

he tries very hard to rub the negation out by some very peculiar reasoning, still, as there was no positive evidence whatever that his "diploccus" was the cause of Texas fever, it can not be seen how Salmon could possibly have said in an early part of the same report, upon the same subject, that:

"*If it had not been* for our private laboratory, the scientific investigation, for the year, with this disease, must of necessity have been a failure, as such investigations had been in the past." Ibid., p. 33.

Which is as much as to say, that his (Salmon's) investigations have not "been a failure." However, further on in the same report he admits, as in other places, that "they have been a failure," in the following words:

"We are not yet certain as to the germ which is responsible for this trouble, and much less do we know anything of its habits of life." p. 43.

And still again:

"The first step towards this solution is evidently a thorough study of the virus; the determination, if the diplococci which I have discovered in this virus are the essential agents of the disease." Ibid., p. 43.

On another page comes another mass of inconsistencies, where he says:

"And so, if the inoculability of Texas fever had not been discovered and demonstrated during the past two summers, we should still be in doubt as to the possibility of determining the connection of the diplococcus of the speen with the etiology of this plague.

"The disease may be produced by inoculation with splenic pulp. This is one point gained and a most important one." Report 1883, p. 44.

How in the world Salmon could have written such positive assertions in the same report where he has said:

"*What conclusions are we to draw from these experiments?*

"*They are certainly negative.*" Ibid., p. 38.

Dr. H. J. Detmers also made some observations upon the southern cattle plague, which may be found in the agricultural reports of 1880, '81, '84, and says that he found a "bacillus" in the tissues; but as he gives us no other evidence of its connection with the disease, we can feel assured that he failed to make out any case for its etiological connection with the southern cattle plague.

THE TRUE GERM OF THE SOUTHERN CATTLE PLAGUE.

We have said that the chief cause of the infection of our northern pastures must be sought in the manure of southern or diseased northern cattle. It now remains for us to offer the proof of that statement.

In order to prove that it is the manure of infected cattle which lodges the germs of the southern cattle plague, we must first find the germ. Has anybody found it? To which I answer that there has, and that the honor belongs entirely to Nebraska, as well as does that of completely connecting the germ of swine plague with that disease and discovering the true nature of that pest.

How may we know that we have discovered the germ in any specific disease? In order to make such an assertion the following conditions should be fulfilled when possible:

First—In the tissues of animals ill with a specific disease must, in each case examined, be found the same germ.

Second—This germ must be cultivated, free from every other germ, in some of the artificial media.

Third—It must be shown that the germ in question has pathogenetic (disease producing) qualities by inoculating animals and killing them thereby.

These three conditions have been fulfilled. The germ of the southern cattle plague has been found in the blood, the gall, the urine, the liver, spleen, and kidneys of every animal that we have made an autopsy on that was diseased. These germs have also been cultivated in an absolutely pure form upon and in artificial media. Gophers, or ground squirrels, have been inoculated with such cultivations, and died from the effects, and the same germ found in their blood and tissues and in sections made from their organs. Cultivations from the same have also been made, invariably showing the same germ as that got from the cattle.

These results, however, do not show that this was the germ of the southern cattle plague. They only show that a germ was found in the tissues of diseased animals that had fatal disease-producing properties.

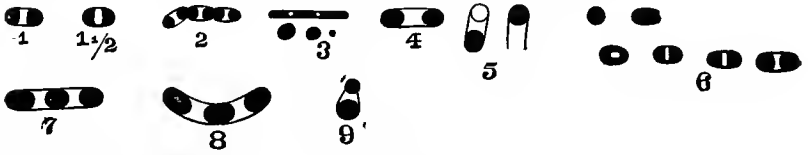
How, then, can we tell that it is the specific germ of the southern cattle plague?

To be able to affirm this fact positively cattle must be inoculated,

as the ground squirrels were, with unquestionably pure cultivations, and the southern cattle plague produced in those cattle, and the same germ found in their tissues and cultivated from them. We have done this, and can demonstrate the entire series of facts by cultures and microscopic specimens of the tissues.

Hence the germ of the southern cattle plague has been discovered, and I think that I may be pardoned the egotism of claiming this to be the first occasion in the history of American medicine that not only one but several germ diseases of animal life have been traced out, and their origin placed upon an impregnable scientific basis.*

MORPHO-BIOLOGICAL CHARACTERISTICS OF THE GERMS OF THE SOUTHERN CATTLE PLAGUE; THE AMERICAN SWINE PLAGUE; AND THE "CORN-STALK DISEASE."



Diagrammatic representation of the phases of development of the ovoid germs of extra-organismal septicæmiæ.

Although the micro-etiological organism of the American swine plague has been described in my report on that disease, and some points touched upon that will not find mention here, still I may be pardoned, in this place, the repetition of details with regard to these organisms, on account of their close relation and resemblance to one another, and the light which the investigation of these germs throws on that of the yellow fever.

These micro-organisms are neither to be classed with micrococci or bacilli.† They are not round objects like the former, or rods like the latter. They belong to that intermediate group, to which, for convenience sake, patho-bacteriologists are beginning to give the name

* The observations made in 1887 have been again confirmed in 1888, by the examination of blood and tissues from diseased cattle in outbreaks in Nebraska, and also from some of the cattle infected in the experiments made in the Chicago stock yards under the auspices of the Illinois Live Stock Commission. For the careful selection and punctual delivery of this material, I desire to express my thanks to Prof. R. J. Withers, President of the Chicago Veterinary School.

† For practical reasons I still think it justifiable to adhere to this opinion. Although under many conditions these organisms do assume a true rod-like form, and even develop into threads made up of rod-like segments, still in their mature and characteristic form they are invariably ovoid and belted as described.

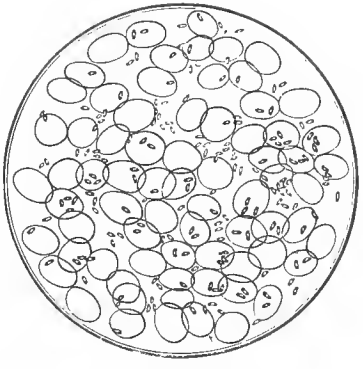
"bacteria," which, while not perhaps a scientific classification, has many practical reasons in its favor. Their longitudinal diameter is about twice that of their transverse. They are ovoid. Their ends are rounded. If an endeavor be made to differentiate these germs from one another by a microscopical examination, we shall find it impossible. They are approximately of the same size and shape. Fresh specimens of them both will not differ so much in dimensions —under the microscope—as old cultures of either will from fresh ones, or different individuals in the same old cultures. They are about $\frac{1}{6}$ the transverse diameter of a red blood cell in length. In one way, however, they can be easily differentiated by microscopic examination.

The swine-plague germ has a far sharper chemical affinity (its poles) for the blue and violet tinctions than that of the southern cattle plague, and the latter possesses a special affinity for fuchsin, while the former does not.

Whatever the tinction used, if applied lege-artis, the ends, or poles, of these micro-organisms will show a greater specific affinity for the coloring material; while the middle portion of the body has far less, unless the exposure to the tinction is unduly or longer pushed, when this portion of the body will eventually color. The capsule of these germs seems to be composed of the same material as the ends, as it colors in the same manner, thus presenting a delicate line of colored substance connecting the two colored coccoid ends, or poles. The most practical illustration which can be given of the microscopic appearance of these two organisms is to take a small white bean and paint both of its ends and two of its sides blue or red, leaving the middle portion unpainted. Looking down upon such a bean would give the observer an almost exact picture of these micro-organisms. (See opposite page.)

Like the genuine and only germ of the American swine plague, the micro-organism of the southern cattle plague is motile in fluid cultivating media when observed microscopically, as well as in the blood serum of diseased animals. The movements of the latter are, however, less rapid, or active, than those of the former organism.

These organisms have not the active shooting or straight-out movement of many others; but they change their locality in the field, they



Mature form of the Germs of the Southern Cattle Plague, the Swine Plague, the Corn-Stalk Disease, and Yellow Fever. x 800.

turn over and over, rise and fall in the drop, move sideways, and when two are united together, twist in various directions, in their endeavors to separate, just as we try to break a rather tough stick in two.

In my descriptions of the micro-organism of the American swine plague, I have called attention to the great morphological variations which it undergoes in completing its full cycle of development. These are its morpho-vegetative phenomena. To one entirely unaccustomed to observing them the first appearance of a microscopic specimen of a cultivation of these germs, more especially an old one, would prove very puzzling indeed. In fact, the novice would very often conclude that his culture had become polluted by micrococci, so plentifully are these objects apparently represented. They simply represent a vegetative or embryonal period in the development of this class of micro-etiological organisms. The views of Hüppe, a very eminent German authority, are very misleading upon this point. He describes, or classifies, this class of germs as "micrococci." It would be equally logical, however, to call an ovum a man, or an apple seed an apple tree. It is far more practical for the patho-bacteriologist to stick to the name "cocci" for all round objects—not spores—which have equal diameters in their mature form, and which color diffusely, and to call these ovoid organisms bacteria, where the longitudinal diameter is not much more than double the transverse. As to bacilli, spirilli, etc., there need be no dispute, so plain are their morpho-characteristics.

The mature micro-etiological organism of the American swine and southern cattle plague has been described above—Fig. 1— as resembling a white bean with its ends and sides so painted as to leave the middle portion of the body untouched, as we look down upon it. That is the picture which the eye of the observer generally receives; but a more exact inspection of a stained covering glass specimen will show that the above is not always the appearance presented to the eye, even by the mature germ. Many specimens may be seen in which the white belt does not extend entirely across the object and there will be more uncolored substance upon one side than the other (Fig. 1½). At first I mistook this appearance for the accumulation of the uncolored substance in this way during the process of its secretion by the pole-ends, which I take to be the method by which this non-coloring material is produced. The whole organism is surrounded by a cap-

sule, but naturally we do not see that portion covering the pole-ends, as it colors at the same time with them. The question now arises, if the whole capsule colors why do we only see evidence of the same on the sides and not on the part presented uppermost to the eye, which appears uncolored?

Whether or not this appearance of more color in the capsule upon one side or the other is due to the action of the heat in drying the covering glasses, is more than I can say, but the reason that we only see the capsule colored on the sides, under appropriate treatment is very evident. It is an optical phenomenon!

The whole capsule colors exactly alike, with the above exception, but being so extremely delicate we do not perceive the color in that portion presented to the eye by the middle of the object, on account of its thinness, but in looking at the side we look through more material and hence see more color, just as in looking through a glass slide or piece of window glass, it appears clear, but if we look through more volume of glass, by looking at its edge, we perceive a more or less greenish shade, according to the quality of the glass.

Again, we may see two or three of these organisms united together, all presenting the normal characteristics of full maturity (Fig. 2). In general they appear either singly or in pairs, though in certain media they have a tendency to form long segmented threads, as well as in some organs. This is more true of the Swine Plague organism than the others, so far as my observation has at present extended. In very old cultures these micro-organisms become thinner, more rod-like, and color more diffusely with the same degree of exposure to the tinction, and the white substance is either not visible at all or is very faint (Fig. 3). Again, such old cultures are very replete in apparent micrococci of various dimensions, which might lead one into the error of assuming that his cultures had become polluted. I call this last condition that of coccoid degeneration (Fig. 3); or we may see unusually long objects, the longitudinal diameter of which being twice or three times that of the mature germ, the white or uncolored substance occupying a corresponding extensive amount of space, while the refracting or colored pole-ends may be somewhat larger or of the same size of those of the mature object. This condition represents the first step in the development of these organisms: that is, they become longer and more of the white non-refracting material is secreted (Fig. 4).

The next step in the process of vegetative development is the separation of one of the pole or coccoid ends, which becomes free and for a moment is exactly round like a coccus; and, as in a hanging drop culture (to which I always add a very slight amount of an aqueous coloring solution), one will naturally see a very large number of these coccoid objects, on account of the fact that each individual germ present is continually going through the same process of multiplication. Here, again, we may see a phenomenon that might be misleading: one of the coccoid ends having been separated, the other still remains attached to the white non-refracting material; and, as evidence that the refracting pole-ends have a greater degree of specific gravity, as well as chemical composition, we may see in the continual tumbling about and turning over and over of these objects, a white, round, or nearly so, colorless, non-refracting object, or numbers of the same. When the micro-organisms in such a hanging drop culture have died from want of nourishment we may see a large number of these objects, which can be easily mistaken for spores. But if we inoculate a new hanging drop culture from the same material used to prepare the former, it will be found impossible to fall into any such serious error. It will be easily seen, then, that these uncolored refracting points keep continually going out of sight, their place being taken by the non-refracting point still attached to the other end of the white substance. By watching one and the same organism in its continuous turning over and over, first one appearance and then the other will be presented to the eye, until the second coccoid end has become detached (Fig. 5).

What becomes of this colorless refracting middle piece?

I do not know!

To my mind this material within the capsule which does not readily take up the tinction is a fluid, and it seems to me as if this fluid became free with the separation of both pole-ends, and that the capsule underwent dissolution at the same time. That this white belt (in the complete organism) does not represent a spore condition or have any relation whatever to spores, is entirely beyond all question, as I have now searched diligently for spores for over a year in both old and new cultures of the swine plague germ, and in others made at all kinds of temperatures within the bio-limits of these organisms, but in vain.

We left our studies with the mature object proliferated into its first distinct stage of vegetative differentation. We had two coccoid objects before us; that is, two round objects, their diameters being the same in any direction. If colored, they color throughout; that is, diffusely. Were these objects to remain in this condition, they would be indeed micrococci. They do not, however. They almost immediately begin to increase in a longitudinal direction, but in this condition they still stain diffusely.

In my first description of the swine plauge germ, I said that the next biological phenomenon was the appearance of a delicate white line separating this ovoid object into two halves. The above, while not exactly an erroneous description, is certainly anticipated by another phenomenon in the evolutional development of this coccoid diffusely coloring object into the mature germ of any of this class of diseases. That this white non-coloring substance is a secretion of the two pole or coccoid ends of these "belted" germs, is beyond all question, as well as that it has a different chemical composition. These two facts, when taken together with the previously stated one, that the white substance almost, if not instantly, disappears from view the moment both of the coccoid—pole—ends have become shed off, segmented, leads directly to the following hypothesis:

May not this white substance constitute, aside from the capsule, the ptomaine, or essential poisonous pathogenetic principle, in connection with these "belted" septicæmic germs, and may not this process of the immediate dissolution of this white substance be the means by which this ptomaine gets into solution, and thus permeates the fluid cultivating media and the blood?

To my mind, this supposition is worthy of consideration. The fact that we can find no evidence of the development of permanent spores by these germs, and that this white substance is a secretion of the pole-ends, goes largely to support this hypothesis.

The phenomenon above spoken of as anticipating the formation of the segmenting white line which separates the two darker portions of these organisms is, that this white substance first appears in the center of the body of the dense, dark ovoid object as the minutest of white specks, which gradually increases in size and quantity, and extends across the entire object, the white line being at first broader in the middle but gradually widening until it completely and clearly sepa-

rates the two pole (coccoid) ends, and the mature object is again presented to our view. (Fig. 6.)

We have thus described the normal or general cycle of development of the micro-etiological organisms of the genuine swine plague, the (American) southern cattle plague, hen cholera, the German "Wild seuche," (of deer, swine, and cattle,) rabbit septicæmia, and last but not the least in importance, the micro-organismal cause of the corn-stalk disease in cattle,* all of which diseases are caused by a member of this class of "belted" germs, and should be classed as extra-organismal, local, or land septicæmiæ. It seems to me (as will be shown later) that the germ of yellow fever, as well as the disease itself, should also come into this group.

MORPHO-BIOLOGICAL CORRESPONDENCE BETWEEN TWO OR MORE MICRO-ETIOLOGICAL ORGANISMS NOT SUFFICIENT GROUNDS FOR PRONOUNCING THE DISEASES WITH WHICH THEY ARE CONNECTED IDENTICAL.

The details of this discussion will be found in my report upon the swine plague. It is necessary, however, to touch upon the essential points here also. As is there shown, Hueppe asserts that the European diseases previously mentioned as being caused by a member of this group of belted, ovoid germs, viz., the "German Schweine Seuche, Huhne Cholera, Kaninchen Septikæmie, und Wild Seuche," (which generalization would also include our American swine plague, southern cattle plague, and yellow fever, and the corn stalk disease,) are all one and the same disease, because their micro-etiological organisms have the same form, the same size, the same belted appearance, and because they all grow nearly alike in boullon, upon agar-agar, and in beef-infusion gelatine.

German authorities are somewhat contradictory as to the deportment of these germs upon potatoes. Loeffler says the one seen by him in 1882 did not grow on potatoes; but the fact is, the Loeffler-Schutz organism does grow on potatoes, as I am assured by eminent German authorities.

I am sorry to say that I cannot agree with Hueppe's attempt at generalization in this class of diseases. According to my opinion, it may be axiomatically asserted that the most complete morphological re-

* See Bulletin No. 8, printed next in this Report.

semblance, and exact morpho-biological resemblances, in or upon any artificial media, are not sufficient grounds for such generalization in classification of these diseases.

To all beginners in the work of patho-bacteriology, and to older hands as well, I most dogmatically assert that there is but one factor in the biology of micro-etiological organisms which can decide whether two apparently alike germs are one and the same object when derived from two distinct diseases of animal life. That factor is a physio-chemico-biological one! The character of the ptomaine produced decides it. Both germs must produce the same disease in both species of animals, and the same clinical and pathological phenomena which occur in the same disease and in the same species of animals under natural conditions, when healthy animals of the given species are inoculated with artificial cultivations of the germs in question.

Our experiences here do not conform to Hueppe's hypothesis!

The American swine, the southern cattle plague, the yellow fever, and the corn-stalk disease, should, according to Hueppe, be identical diseases with those mentioned as such by him in Germany; because, according to his conditions, the germs are identical in appearance. Hueppe's entire argument is completely nullified by the following facts:

First—There is no southern cattle plague or corn-stalk disease known in Europe at present.

Second—Cattle and swine run together in this country, and one or the other may have its specific disease and yet the other species will never become ill, even from the closest contact with the diseased individuals.

Third—The same is true of hen cholera and swine plague in this country. Hens can feed on hogs dead from swine plague, from the ground polluted with their discharges, even picking out grain from the same, and still remain well; and the same is true of the hogs with regard to hen cholera, southern cattle plague, and the corn-stalk disease.

Hence, no matter how much germs may resemble each other when microscopically examined, or even in or on many cultivating media, they fail in the one great factor necessary to make the diseases produced by them identical.

Fourth—Human beings do not have the yellow fever at the same time cattle have the southern cattle plague in the same locality. They

do not have the same physiological chemical attribute with regard to a given something produced which invariably decides the pathogenic results produced by a given germ. Notwithstanding the latter fact these diseases have a very close relation to one another.

They are all extra-organismal, local, land septicæmiæ. Each one, however, has something peculiar about it that prevents it from being identical with the others, aside from any action of the germ itself. It is this : each species of animals in which they produce a specific disease has some unknown constitutional idiosyncrasy which renders its members susceptible to the action of a given germ, and each of these germs has some peculiar unknown biological idiosyncrasy by which alone it infects naturally but a given species of animal life. These two factors together can alone decide the identical question. What we can do artificially by the inoculation of other animals than the disease occurs in naturally, has no necessary relation to the question whatever.

To return to our subject.

There are, however, other phases in the development of these germs of a bio-morphological character. For instance, as already said, we may see two or three individuals, of the mature type, united together (Fig. 2), or we may find two apparently mature organisms enclosed in a common capsule, the two medial dark points or poles being in such close apposition that no line of demarkation or indentation of the capsule can be seen at this point, the whole outer surface being smooth (Fig. 7); on the other hand the two lateral ends or free poles are separated by the normal quantity of white non-colorable substance. Again, these diplo-bacteria may assume a curved or sausage shape, which we may sometimes see intimated in the single organisms (mature—Fig. 8). At other times, though not very frequently, the germ may appear in a nearly normal form, but one pole-(coccoid) end will be semi-segmented from its appositional end of the white substance by a constriction of the same at its line of attachment with the pole-end (Fig. 9). This end will then be smaller than the opposite pole, thus giving a sort of pear shape to the entire organism; the small pole-end is soon dropped, however, and becomes momentarily a free coccoid, and goes through the cycle of morpho-development already described; the same occurs with the other pole-end. This concludes my observations, of the micro-morphological phases presented by these

two etiological organisms in the course of their development. There may be some minor phenomena that have escaped my attention, but I am very sure that I have described all the essential points.

EXAMINATION OF ORGANS OF ANIMALS DISEASED WITH SOUTHERN CATTLE PLAGUE FOR MICRO-ORGANISMS.

Spleen, Cattle.—As mentioned in the text, and greatly to my surprise, I have been unsuccessful in obtaining cultures of the germ of of this disease from the spleen of diseased animals, notwithstanding the fact that the blood from the heart and larger vessels was microscopically found to be replete with them, and cultures successfully obtained from this medium. This failure to obtain such cultures from the spleen must, it seems to me, have been due to some error in technique — what, I do not know, but perhaps too hot a wire. At least, after having so signally failed in several instances, I afterward gave my attention entirely to the liver and kidneys for the purpose of obtaining cultures, having always been as positively successful in this regard with these organs as I have been unsuccessful with the spleen.

Microscopic examination of stained sections of the spleen shows this organ to contain a plentiful representation of the etiological moment described in these pages as the cause of the southern cattle plague, and so far as can be seen, isolated individuals and not connected together. It is unnecessary to again call attention to the various phases in which this organism presents itself in the tissues of diseased animals, as it has been mentioned several times in different parts of this report.

In the liver they are thickly represented, lying mostly loose between the cells, but sometimes enclosed in a round cell; again, in the capillaries, but here also as single individuals.

In the lymph-glands before us some care must be taken not to mistake a fine granulous pigment, especially when the granules are slightly separated from each other, thus leaving a clear space between them which presents a picture somewhat resembling the pole ends and belted appearance of the micro-etiological organism of this disease. A mistake, however, is entirely unnecessary, the granules of pigment being larger, their outlines irregularly defined, and of a greenish yellow color. I would not call attention to these facts had it not been that really good microscopic diagnosticians have mistaken

these granules in these very specimens for micro-organisms, and, being all alike, for one and the same kind. The real germ, however, is much smaller, its pole ends sharply outlined, and middle-piece clear and distinct when an appropriately-situated object is under the eye. Here, too, they are present as isolated individuals. They are also to be seen in large numbers in the lungs, especially in the vessels of the interstitial spaces, but also scattered through the substance. It should be mentioned that this tissue was taken from a hypostatic lung, and not from a consolidated one.

The same organisms are also present in the same manner in the spleen, liver, and kidneys of the steer inoculated with the pure cultures, (the details of which experiment are given in another part of this report,) as well as in the sections of the kidney, lung, liver, and spleen of one of the numerous ground-squirrels which have been used for experimentation.

THE GERM OF THE YELLOW FEVER.

So far as my personal experiences extend, and the literature at my command will permit of my expressing an opinion, while a vast amount has been written upon this disease, there does not seem to be one of the great human pests which has been so unsatisfactorily studied, from an exact and scientific point of view. While the clinical reports will answer, and the empirical-epidemiological phenomena are passingly satisfactory, one looks in vain for an exact and detailed description of the patho-anatomical lesions. Search where we may in all the leading works; through all the special journals upon such subjects; overlook as we may the reports or papers of the so-called, and much lauded scientists who have been especially deputed to investigate this disease, *not one single descriptive autopsy is on record.* In fact a competent pathologist, with a knowledge of kindred diseases, and well versed in their clinical phenomena, could easily write a more accurate and detailed description of the anatomical lesions in yellow fever than any one of these observers (?) has given us to date. It matters not what persons may think of the writer's methods, or what adverse opinions they may form of his character, he does not hesitate to say that any and every unbiased pathologist must agree with him that, judged by the evidence as given by themselves, and as it exists in printed works in England, Ger-

many, France, or the United States, those persons who have been engaged to investigate the yellow fever have given the strongest testimony possible of their utter incapacity and unfitness for the responsibilities conferred upon them. They do not give evidence that they even know how to approach a question of this kind. They imagine themselves bacteriologists;—some because they have edited a book written by another, and found some of the fossilized editors of semi-petrified American medical journals willing to praise them, and because of this have finally been led to believe themselves authorities upon a subject their very work has demonstrated their utter incapability of investigating.

Whoever carefully follows my endeavors to free scientific research from the octopus-like strangulation which it is suffering under in this country on account of the untoward influences underlying American political life, must be aware that no matter how severe my censures may be, they are supported by not only uncontrovertible evidence, but by testimony which is well known to be reliable by the unbiased public, and the intelligent part of the American medical profession.

In an address before the Quarantine Conference at Montgomery, Ala., March 5, 1889, upon "Hunting Yellow Fever Germs,"* Dr. Sternberg admits that he has been hunting for the germ of yellow fever for ten years, and that he has been and still is a most unsuccessful hunter, as follows:

"You are aware that my first effort to solve the etiology of yellow fever was made ten years ago. As a member of the Havana Yellow Fever Commission of the National Board of Health, I had an opportunity to make researches which, in advance of the effort, I fondly hoped might lead to demonstrations alike creditable to American science and useful as a basis for preventive and curative measures in this pestilential malady. * * * It was therefore with the deepest interest as well as with the strongest hopes of success that I went to an endemic focus of the disease to search for the yellow fever germ."

In a previous part of the same address the speaker said: "But I must announce to you, in advance, that there is no satisfactory evidence that any one of these micro-organisms is the veritable infectious agent in the disease under consideration."

The reader will now please turn with me to that portion of the address where the author is speaking of what he did as a member of

*The Medical News: Philadelphia, March 9, 1889.

that "Yellow Fever Commission," and I especially desire, and emphasize the fact, that the reader will notice that the language to be quoted bears direct relation to Dr. Sternberg's work as a member of that commission, and that Dr. Sternberg himself says now, 1889, that he was fitted to engage upon the work, as follows:

"I was" [then 1879] "familiar with the most approved methods of mounting and staining micro-organisms, and was provided with the best high-power objectives that could be procured. * * * *Not only did I feel that I was equipped for the recognition of any micro-organism which might prove to be present in the blood, but I was prepared to photograph it, and thus to show to others what I might see in the blood drawn from the circulation of yellow-fever patients.*"

Dr. Sternberg undoubtedly believes all which he said of himself in this Montgomery, Ala., address, 1889, was equally true of himself when a member of the "Havana Yellow Fever Commission" in 1879; but he seems to have forgotten that some years previously he edited a book entitled "Bacteria," Magnen, 2d edition, 1884, in which is printed, page 420:

"The writer's personal investigations are recorded in the 'Preliminary Report of the Havana Yellow Fever Commission of the National Board of Health." * * * *

Of his fitness to do the work at that time Dr. Sternberg said:

"Evidently an extended acquaintance with the bacterial organisms found during life and after death in the bodies of persons not suffering from yellow fever, *and familiarity with the most approved methods of isolating and cultivating these organisms, would have been of great advantage to the investigator. But this preliminary knowledge and special training was of the most imperfect character.*"

Certainly further comment is entirely unnecessary after such a display as that by the patho-bacteriologist of the "Yellow Fever Investigating Commission," supported by the Government of the United States.

Let us now turn to the investigations in search of the germ of the yellow fever, and begin by considering those of Freire, who read a paper giving what I suppose can be looked upon as the details of his work, before the "Section of Public and International Hygiene of the International Medical Congress, Washington, 1887," and published in abstract.* I will not, however, use that paper, but will insert a

* Medical News, Phil., Sept, 17, 1887.

summary furnished me by Dr. John S. Billings, and published in the first edition of this report, as it is more condensed, and has the advantage of being in the exact words of the author.

"RÉSUMÉ DE L'EVOLUTION DU CRYPTOCOCCUS XANTHOGENICUS."

Milieu dans lequel il vit	"Dans le sang, dans le liquide du vomissement, dans le cerveau, dans les muscles, dans les organes parenchymateux, en général dans tous less tissus et humeurs de l'économie et dans les liquides de culture albumineux."
Mode de croissance	"Ils commencement par de petits points noirs, qui passent à l'état cellules noudes, bordées de gris ou de noir, réfractants fortement la lumière, quelquefois irisées; granuleuses à l'intérieur lorsqu'elles sont mûres, contenants, du pigment jaune et verdâtre."
Mode de reproduction	"Les cellules mûres se déchirent: 1° en différents points en même temps (c'est le cas le plus fréquent); 2° en un seul point, les spores sortant par un seul orifice (rare); 3° par une section circulaire, la cellule prenant la forme d'un pyxide? (Rare.)"
Disposition des spores, des lambeaux et du pigment	"Tantôt les spores se répandent sans ordre, tantôt ils adhérent au pigment sans disposition réguliére, tantôt ils adhérent à eu même affectant différentes figures, comme celle d'une poire, d'une pomme de pin, d'un ananas. Les lambeaux provenant de la déseggregation des cellules, forment divers amas amorphes, blancs, noirs on verdâtres."

"RÉSUMÉ OF THE EVOLUTION OF THE CRYPTOCOCCUS XANTHOGENICUS.

Media in which it lives.	"In the blood, vomited fluids, the brain, muscles, and in the parenchymatous organs; in general in all the tissues and humors of the economy, and in albuminous cultivating media."
Mode of growth, (appearance.)	"They commence as small black points, which pass to a state of round cells, having a gray or black border which is very refracting; they are granulated, when mature, in their interior, containing a yellow or green pigment."
Method of reproduction.	"The mature cells divide themselves as follows: "1. At different points at the same time—which is most frequent. "2. Into a single point, the spore passing out by a single orifice—rare—(! !—B.) "3. By a circular section, the cell taking the form of a 'pyxide capsule'—rare—(! ! !—B.)"

Disposition of the spores, of the segments, and of the pigment.

"Sometimes the spores disperse themselves without order; sometimes they adhere to the pigment without any regular disposition; sometimes they adhere to each other, forming different figures, resembling a pear, a pine cone, or a pine-apple. The segments proceeding from the disintegration of the cells form divers amorphous clusters of a white, black, or green color."

So much for Freire's description. That it will not apply to any known form of bacterial life need scarcely be called attention to. That Freire, like Pasteur, is ill-fitted to enter upon such studies, in the spirit of exact pathology, is self-evident, but still there is an important statement in the abstract noticed above, which it will not do to overlook.

Under the heading, "Inoculability of the Amarillus Microbe," Freire says: "I have made a great number of inoculation experiments upon animals in order to prove the transmissibility of the disease." "*I have remarked that not only do inoculations made, directly or indirectly, with the blood and with virulent cultures, kill rabbits and Guinea pigs in from two to ten days*——"

That is the point! Here is a positive statement, which in the want of positive contradictory evidence, it will not do to deny, though we do not have the full particulars, that not only in the blood, but in cultures made from materials from yellow fever patients, there is a something with virulent properties, though we cannot distinguish any specific characteristics by which to recognize that object in Freire's descriptions. This does not militate against the fact, which is exactly on a par with Pasteur's results and descriptions in the Rouget of the hog, in which he said a coccus was the germ, but which Schutz showed to be a bacillus, which object was in Pasteur's virus also, but overlooked by him. I say, we cannot overlook a statement like the above.

Scarcely any one can doubt that the yellow fever is a septicæmia, a blood poison, and I think all competent persons will agree also in its being of an extra-organismal origin — that is, an exogenous disease. Being this, it is an incomprehensible phenomenon why both Sternberg* and Gibier† should be so unsuccessful in discovering any micro-organism in the blood, at least in some cases, as my own inves-

*Med. News, l. c.

†Med. News, January 26, 1889.

tigations have conclusively demonstrated not only the presence of such, but of one and the same organism in the coagulated blood filling small vessels in sections of the liver and kidneys, from seven different individuals, each of which is guaranteed as having undoubtedly died from the yellow fever; and from the liver of an eighth individual, the genuineness of the diagnosis being also unquestionable in this case as well.

Sternberg says: (l. c.:) "Ninety-eight specimens from forty-one undoubted cases of yellow fever were carefully studied, and one hundred and five photographs were made, which showed satisfactorily everything demonstrable by the microscope. No micro-organism was discovered." His other and later attempts have also been "negative as regards this tissue.

These "carefully studied" cases were made when Dr. Sternberg was member of that "Havana Yellow Fever Commission," and we have seen that when the facts as to his qualifications (1879) were still fresh in his mind, though they were completely dispelled later, 1889, the necessary "preliminary knowledge and special training was of a most imperfect character,"* which he admitted even as late as 1884; and hence we may feel justified in assuming that the requisite skill to demonstrate this organism has not yet been acquired by this investigator. All attempts at cultures from the blood have also been unsuccessful in the hands of Sternberg and Gibier, though said to have been successful by Freire and Lacerda. Exactly similar assertions have emanated from the bi-bacterial chief of the swine-plague investigations in Washington regarding any micro-organism in the blood of cattle diseased with the southern cattle plague, and very lately a most reliable gentleman has personally told me that they could not find any germ in the blood of Texas diseased cattle, but had found one like mine in the contents of the intestines. Any one who should be here can be shown the original specimens from the blood of cattle which died of this disease in several outbreaks, dating back to the first investigated, in 1887, and in each one of them can see absolutely pure conditions of the germ of that disease.

Dr. Paul Gibier was sent by the French Government to study this disease during the late eruption in the Southern States, and contributes a paper on the "Yellow Fever: An Experimental Study of its Eti-

* Magnen — Sternberg.

ology," (Medical News, l. c.,) which is absolutely worthless except to show what the author did not do.

In the first place, he is inclined to attribute the black vomit in peracute cases of the yellow fever, to a pigment secreted by a germ which he thinks specific because "its biological qualities are entirely in accord with the physiology of yellow fever itself, and in certain conditions it blackens the bodies it comes in contact with, so much so that some liquid cultures have the appearance of black vomit," though he immediately qualifies this remark by saying, "I am far from pretending, although it has been so claimed, the last character, no matter how suggestive it may be, is pathognomonic." Then he admits "it does not always appear." Sternberg says, (Medical News, l. c.,) "The bacillus of Gibier I have only isolated in three cases: * * * I have never observed in my cultures the black pigment which, according to Gibier, is produced during the development of this bacillus, and am at a loss to understand this discrepancy in our observations."

If Gibier can be trusted, and his bacillus does produce black pigment, then Sternberg cannot be correct in asserting that he has met the "bacillus of Gibier," for on such a simple question one or the other must be correct, and the one who errs incompetent. Either the bacillus is melanogenetic or it is not. On the other hand, I agree with Sternberg when he says, "So far as the pigment in black vomit is concerned, I have no doubt that it is of hæmic origin; and not only because of his evidence that red blood cells are present in the vomitus, but because exactly similar conditions may be seen in occasional cases of the southern cattle plague (even though the animals do not vomit,) and occasionally in swine plague.

It would have been well had Gibier given American investigators a description of an organism to which he attributes so much importance as to claim, "I think I am right in saying that the presumption that this bacillus is the cause of yellow fever tends to become a certainty."

But he gives us no evidence whatever in support of such a statement. On the contrary, all he has to say is directly opposed to any such conclusion. It has not even collateral evidence in its favor.

Gibier continues: "Besides, I must notice that in cases in which autopsy took place early after death, the blood, liver, spleen, and kidneys have been constantly found free from microbes."

This is simply an assertion to be taken *cum grano salis.* It is no more than saying, with Sternberg, "I could not find them," or words to that effect.

But to go on with our French authority, who says:

"This fact strengthens the theory which I have supported, viz., that yellow fever is an intestinal affection.

"However two objections may be presented which I must answer:

"1. How is it that the microbe, supposed to be pathogenic, is not found in every case after death, and if it has disappeared, how shall we explain the persistence and the aggravation of the accidents?

"2. If the yellow fever is a disease the germ of which grows exclusively in the intestines, how shall we explain the albuminuria?"

Earlier in my remarks I have spoken of the lamentable pathological inability of these investigators, as evinced by the total absence of one detailed necroscopical description, and as further evidence in the same direction, I point to the second question proposed by Gibier.

What should cause the albuminuria but the same factors which cause this phenomenon in many cases of acute diseases of the character of the yellow fever? (In fact, every one of these septicæmiæ is accompanied by albuminuria.) What but *the extreme fever and sequential disturbances of the circulation, and parenchymatous changes in the kidneys in consequence thereof?*

The kidneys are also infested by micro-organisms in this disease, Gibier, Sternberg et al to the contrary.

Gibier is undoubtedly right when he says that "the yellow fever is an intestine infection;" but further than this I decline to follow him. Were the respiratory tract the way by which these germs gained access to the human organism, we should have received some intimation of pulmonary lesions; but as we have not, the only possible way of infection must be by the digestive tract. Exactly the same thing occurs in the southern cattle plague; but here we are very much in advance of these observers, for we have demonstrated in all necessary ways the presence of the specific germ in the intestinal contents, as well as in the blood, and organs. In fact, it will do these gentlemen no harm, and we advise it also to the U. S. Government, as yellow fever appears to be so far beyond their limited abilities at present, to take a little practice in a disease where the way has been opened for them, and study the southern cattle plague. After they have done

that satisfactorily, they may be able to do something with its twin-sister, the yellow fever.

Anyone who has had long experience in the experimental study of this class of diseases caused by a member of the ovoid-belted germs, but more particularly with such as the southern cattle plague and yellow fever, in which infection invariably occurs *via* the digestive tract, will be soon struck by the very great variation in the number of germs found coursing in the blood and present in the organs in different individuals. I think it can be taken to be almost a rule, that the more prolific the infection, the more rapid the multiplication of the germs within the intestine, the greater and more intense will be the irritation of the mucosa, with consequent proliferation of the cells in its follicles and crypts, as well as the greater the amount of ptomaine produced, which, while causing the most acute and severe symptoms by its absorption and dispersion over the organism, is still marked by the few germs, comparatively, which find their way through such an intensely swollen mucosa into the circulation. I have demonstrated this fact by experimentation. The same results can be achieved by the continuous feeding of strongly saturated bouillon in which the germs heve been killed, without one being present in the organism. (See my Swine Plague Report.)

EXAMINATIONS OF PIECES OF ORGANS FROM "UNDOUBTED CASES OF YELLOW FEVER."

As has been remarked elsewhere, no one acquainted with the literature of yellow fever could possibly be long engaged in investigations upon the southern cattle plague of the United States without being most forcibly struck by the many strong points of resemblance between the two diseases. Let me at once most positively assert that *I do not now think, and never have thought, that the two diseases are identical, or that they are one and the same disease;* though, for some unknown reason, several reviewers of the first edition of this work have fallen into such an idea. Notwithstanding the closest resemblance in the lesions, and, as I now again assert, in the morphology of their micro-organismal causes, still any likelihood of these diseases being etiologically identical is absolutely contradicted by the fact that one can, and does, occur in its appropriate species of animal life without the other occurring at the same time in the same locality. The absolute folly

of placing any great weight upon the most exact micro-morphological correspondence in etiological organisms having any evidence in identity-diagnosis, and even the most close biological resemblances, is most conclusively shown in comparisons between the swine plague and the disease treated in the next article, the so-called "corn-stalk disease." Here we have two diseases, one occurring in cattle, the other in swine, the germs of which are so near alike that even the most expert mycologist would not dare to say which was which on a microscopic examination, and which even have many strong points of resemblance in cultures, but, fortunately, also equally strong ones of differentiation; and yet practical experience has conclusively demonstrated that each disease is confined to its own species, and even experiment has demonstrated the impossibility of inoculating the corn-stalk disease in swine. In this case we have again even as strong points of conformity in the lesions induced in swine plague and those in the corn-stalk disease in cattle, as between the southern cattle plague and yellow fever, and yet, as asserted, these diseases are in no way identical, though belonging to the same class—extra-organismal septicæmiæ, and caused by a member of the group of the ovoid-belted germs.

These coincidences between the southern cattle plague and the yellow fever incited so much interest that every endeavor has been taken to procure alcoholic material from "undoubted cases" of yellow fever, and considerable means spent in that direction. I even went so far as to offer my services to the United States Government to go to the infected districts in Florida, the past summer and fall; but the authorities appear to have had such sincere regard for my life and value to the country in other directions that no answer has ever been received from them on the subject. However I was eventually successful in obtaining quite a supply of really reliable material. The first specimens were sent me from Baltimore, by Dr. Geo. M. Sternberg, and arrived at my laboratory in December, 1887, and were noticed in the first edition of this report. They were preceded by a letter from Dr. Sternberg, in which he says, "The material sent you is from an undoubted case of yellow fever."

In his address on "Hunting Yellow Fever Germs," the same observer says: (l. c.:) "In my researches by the method of staining these sections, hardened in alcohol, I have encountered several different micro-organisms, *but no one of these has been found in a series of cases.*

One, the bacillus of Lacerda and Babes, I have found only in material brought from Dr. Lacerda's laboratory, in Brazil, and in two only out of nine cases represented by material from that source."

These are the only cases mentioned by Sternberg in which he found the organism named.

It is somewhat singular what trips specimens of this material have been sent upon. First it went to Babes and Cornil, in Paris; then to Baltimore, again to me in Nebraska, and again I have sent pieces of it to Koch, Hueppe, and Baumgarten, in Germany, as well as to several American observers, as also pieces from the six cases which I received later, to which allusion will soon be made.

Although Dr. Lacerda primarily saw this organism, the first intelligent description of it was given by Babes,* who says:

"The capillaries of the liver and kidneys contain large numbers of jointed filaments. With a Zeiss $\frac{1}{18}$ H. I. one discovers these filaments to be composed of elliptic, cylindrical granules, and united in pairs, or forming small groups, in which they are united by a pale intermediate substance."

The material upon which the above examinations were made was sent to Babes by Dr. Lacerda, of Rio Janeiro.

At a subsequent period, Babes examined other material, sent by another physician, from two patients, but received negative results, as he did with the liver of a third which he examined in connection with Robert Koch. He did not think that his results were positive enough to warrant any conclusive opinion.

In the Comptes Rendus, Tome, CV., P. 289, is an article: "Sur les formes bacteriennes qu' on recontre dans les tissues des individues morts de la fievre jaune," by Mons. J. B. Lacerda, who says:

"In all these preparations, without exception, I have found one bacterium exactly identical with that which Babes has already found in material sent him from here by me."

The reader will observe that Lacerda says, "In all these preparations."

It would seem that Lacerda had given a description of his observations, exactly similar to that which follows, some years previous to the one noted here, for Babes says:

"At the same time (in which B. received material from L.) Lacerda

*Les Bacteries, Coanil-Babes, Paris, 1885, p. 448.

sent a communication to the Academy of Sciences upon the parasites of yellow fever. But according to the drawings attached to this communication, we have convinced ourselves that he has been deceived, and that he has described filaments of vegetable tissue and pigment instead of parasites." Les Bacteries, P. 447.

On the other hand, as Babes has described this organism, as well as illustrated it, in such a manner that we are enabled to recognize it sufficiently for comparison, and as Lacerda asserts that he has also found "in all preparations, without exception, one bacterium, exactly identical with that which Babes has already found," and as even Sternberg admits its existence in the tissue of such subjects, we have very conclusive evidence that it must be the pathogenetic organism of that disease, even without the necessary conclusive experimentation upon man, which must be left out in such cases.

Lacerda's description is as follows:

"These bacteria are dispersed all through the substance of the liver, and are found in clusters in the cells of the organ, and are profusely present in the capillaries of both the bile and blood circulatory system. In the kidneys they are also found in the capillaries, the urinary tubes, and the convolutes of the Malpighian tufts, and in the lymphatic spaces.

"When sections of the liver are colored, the bacteria take up the tinction very unsatisfactorily."

The above does not coincide with my own experiences, as I have found no difficulty in staining the organisms in the liver any more than in the kidneys, of which Lacerda says:

"But in the kidneys, on the contrary, they can be more easily stained, and may then be studied in all their morphological details."

I cannot accept Lacerda's description of these details at all, as they do not describe the single organisms, and such description as he gives of dicho- and trichotomous division of the chains does not occur. But to go on. He says:

"The bacteria invariably present themselves in chains, formed of a series of granules of approximately the same dimensions, being slightly elongated and cylindrical in form, the points of articulation of the granules being very solid, thus opposing the easy disintegration of the chains. When uncolored, the granules are very refractive by reflected light.

"What distinguishes this bacterium from all other known forms is its tendency to constantly present this ramified form! They fre-

quently have trifurcated stems with two lateral branches which leave the parent stem at a common point of origin, where they assume a curved course, the parent stem also developing at the same time. At another time they form long branches, one of which is nearly straight, while the other is somewhat bent, the bent branch being dichotomous!!? In other cases there are two straight branches of approximately the same length, resembling the arms of a pair of open compasses at an acute angle. In still others, the ramifications are placed at right angles, having the appearance or form of a cross. There are others that scarcely have any indications of bifurcation, the two branches being much shorter than the other, and curved in opposite directions. Again, we may see three ramifications of the same length extending from a common center. The center granule of the primary stem, from which these branches extend, is often larger than those of the secondary formations, and presents a spherical rather than a cylindrical form. It is also often the case that the terminal granule of the branches is the largest and most spherical.

"I ought to add that in preparations of the blood I have found forms resembling the same, and I have no doubt that non-ramifying forms found in the blood and tissue are the result of accidental disintegration of the chains."

EXAMINATION OF THE TISSUES FROM DR. LACERDA, SENT BY DR. GEO. M. STERNBERG.

Liver.—Parenchymatous changes quite severe, with an absence of the nuclei in many of the cells; marked encroachment upon the parenchyma in all parts of the organ, due to an increase in the connective tissue, most of which is more or less organized. In the vascular spaces the tissue shows very little cellular structure, the walls of the arteries and ducts being excessively thickened—especially the latter. In the parenchyma itself are to be seen numerous round, almost transparent, hyaline-looking bodies, varying in size from that of the ordinary nucleus of a liver cell to that of the cell itself. Many other cells have been transformed to a granulous detritus.

Kidneys.—Extreme degree of parenchymatous degeneration, the parenchyma of many of the tubes being one mass of granulous detritus, the nuclei being especially conspicuous by their almost entire absence. In other tubes the basement cells present distinctly visible nuclei, while the aforementioned detritus takes up the balance of the lumen. Hyaline casts are to be seen here and there in both transverse and longitudinal sections of the tubes; diffuse centers of round-cell

infiltration, with marked absence of parenchymatous structure in the same, are scattered through the organ.

The Malpighian tufts are marked with the presence of occasional loops, presenting a hyaline, more or less transparent, appearance, characterized by the entire absence of anything resembling a nucleus. The Bowman's capsules are thickened, as well as the walls of the capillaries and membrana propria of the tubes, with an occasional more or less circumscribed round-cell infiltration scattered here and there between them.

Microscopic Examination of the Above Tissues for Germs.—Both smear-preparations and sections are marked by the presence of a most plentiful representation of an ovoid germ with distinct belted appearance and sharply colored pole ends, the normal length being twice as long as wide; some individuals are three or four times longer than wide, this increase in length being entirely due to an increased amount of the aforementioned uncolored substance. In some instances two, three, or four of either of the above-described forms are to be seen attached together, forming short chains. Such a chain is at other times composed of both of these forms united together, there being more of one and sometimes more of the other in the same.

The organisms appeared, as this variety always does, in the sections of organs, some of them end on, when they looked like cocci; beside these would be others lying horizontally, which presented their complete form, the colored pole end and clear center being distinctly visible. In many localities they were united in pairs, while many of the liver cells contained large numbers of them. Here and there one would find a capillary embolus made up of nothing else; here they frequently grew in filaments of considerable length, large clusters of such being present. Occasionally single filaments were to be seen in capillaries which the section had cut horizontal to their course, but in general, except in the embolisms, they were seen in pairs or groups of three members. Capillary embolism was more frequent in the kidney than liver.

No other micro-organism present, notwithstanding numerous sections of the same tissues were subjected to the very many tinctions used in this work.

This result seems to have had a much more convincing effect on my mind than on that of Dr. Sternberg, for, as we see, he still is of the

opinion that "there is no satisfactory evidence that any one of these micro-organisms is the veritable infectious agent in the disease under consideration."* I will not enter upon the discussion of this point at this moment, but will at once proceed to detail observations made upon pieces of material from six other "equally undoubted cases" of yellow fever, which were procured for me in Havana, Cuba.

It required the overhauling of a very large amount of red-tape in order to get this material from Havana, and were it not for the sincere kindness of Dr. John B. Hamilton, the efficient Surgeon General of the United States Marine Hospital Service, at Washington, I should have been unsuccessful. Dr. Hamilton recommended me to Dr. D. M. Burgess, Inspector for the above service at Havana. The difficulties in the way of procuring such material are so well set forth by Dr. Burgess in the following letter that I think it not inappropriate to introduce it here:

"HAVANA, CUBA, July 4, 1888.

"*Dr. John B. Hamilton, Surgeon General M. H. S.:* MY DEAR DOCTOR—I received your communication in which you informed me that you had referred Dr. Frank S. Billings, of Nebraska, to me for pathological material from yellow fever patients, and in due time a letter of instructions came from him. Since then I have been improving every opportunity to get the desired material, but this year it is rather slow work, as very few persons have died outside the military hospital. The law here is to the effect that autopsies shall not be made until eight hours have elapsed after the death of the subject. At that time decomposition in those dying of yellow fever, and of course in hot weather, is well under way, and putrefaction organisms abound everywhere. If one happens to have the necessary influence with the Captain-General, etc., etc., and brings it with him here, autopsies can be got sometimes at the military hospital, for military reasons, etc., pretty soon after death, but these are *exceptional* instances, and by *no means* very common. I have to rely for my supply at present on the charity hospital and any private cases I can pick up. Up to the present I have not lost a person by yellow fever this year, and only one has died in the charity hospital since I received the letter of instructions; that case I captured, and have the proper pathology. The charity hospital is fully three miles from my office, near no public thoroughfare, and has to be approached by coaches over a bad road. Three-fourths of yellow fever cases who die usually select the ungentlemanly and disobliging hour for their departure of somewhere between 12 o'clock and 4 o'clock in the morning.

* "Hunting Yellow Fever Germs," l. c.

"For some reason or other people die slowly when you are waiting for them to do so. They are not at all obliging in that respect. As a matter of fact, few here are devoted to science, and when one begins to look after pathological material *soon after death*, he will encounter less assistance and sympathy and more difficulties and obstructions than perhaps Dr. Billings imagines.

"The sooner after death pathological material is obtained here, the more troublesome and costly. I have succeeded in getting two cases thus far, with prospects of another, but yellow fever is not plenty at all—outside military hospitals. I will work on, however.

"Very respectfully, D. M. BURGESS,
"*San. Inspector M. H. S.*"

The above letter certainly looked discouraging enough, but the old and true saying, "Where there is a will there is a way" was soon exemplified in the case by the receipt of a second letter from Dr. Burgess dated:

"HAVANA, CUBA, December 6, 1888.

"MY DEAR DOCTOR: *I send you by express, on this date, a box containing pathological material from six undoubted cases of yellow fever.* There are, as you requested, pieces of the liver, kidneys, and stomach in each vial. You will see the date of death and time of autopsy, and where such person died, on each bottle."

This material was most beautifully collected and packed, and I desire, herewith, to express my deep gratitude to both Surgeon General Hamilton and Dr. Burgess for their great interest and exceptional kindness.

EXAMINATION OF PIECES OF ORGANS FROM "SIX UNDOUBTED CASES OF YELLOW FEVER FROM HAVANA, CUBA."

CASE 1.—Pedro Marquez, Spaniard; died of yellow fever at Hospital Mercedes, July 31, 1888, at four o'clock A.M. Autopsy at 7:30 o'clock A.M., the same day."

HISTOLOGICAL EXAMINATION OF TISSUES.—*Liver.*—Vessels of the portal circulation engorged and filled with coagulated blood, with more or less leucocytic migration in the vicinity of the capillaries. In those places where the vessels of the portal circulation, the arteries and the gall ducts are situated, the Glisson's capsule is thickened and replete in connective tissue cells. This induration appears to have taken its start from the walls of the arteries. There seems to have been a general irritation of the stroma of the organ, of a mild charac-

ter and recent date, extending from the arterial circulation, there being a slight degree of proliferation throughout this tissue. The parenchymatous cells are swollen, and contain single nuclei more or less enlarged, while others contain two and some three nuclei, the third being generally smaller than the other two. Still other cells are marked by their excessively-swollen bodies, granular appearance, and the entire absence of a nucleus. These cells are sometimes single, but more often collected in groups. Among them, and scattered through the substance of the organ, are a large number of granulous clusters, varying in individual size and extent, but wanting any sharply-defined outlines.

Kidneys.—Vessels of medullary portions decidedly engorged, said condition being also present in individual vessels in the cortical substance, some of which are ruptured, with irregularly extended hemorrhages extending from them into the parenchyma. These extravasations, which vary in size, are dispersed in the immediate subcapsular portion of the cortex. There is a more or less general thickening of the walls of the capillaries throughout the cortical portion, but very slight in degree, the same condition being manifest in the membrana propria of the tubes. There is a general diffuse swelling of the epithelial lining of the tubes, with distension of the same much more marked in some places than others. The parenchyma filling the distended portions of the tubes presents a diffuse granulous appearance, neither the outlines of the individual cells nor their nuclei being visible. In many places, instead of this diffuse mass, the tubes are loosely filled with desquamated cells, many of which do not present any nucleus, while in others the nucleus is of a faded color; in others again it is very prominent; granulous colonies are dispersed among these cells; in other tubes the perepheral layer of cells are marked by the distinctly-visible nuclei, while the center of the tube is taken up by a diffuse granulous mass. Scattered throughout the urinary tubes in all portions of the kidney, in transverse and longitudinal sections, may be seen hyaline casts.

EXAMINATION OF TISSUES FOR MICRO-ORGANISMS.—Cover-glass preparations made from alcoholic material as described later in the text; magnifying power, Leitz' Ocular "O" Oil Immersion "$\frac{1}{20}$." But one micro-organism present and that but sparsely represented. It is ovoid, of about the size of the organisms described in

this report as the cause of the southern cattle plague, when properly treated, and has a distinctly-marked clear center, with equally distinctly-colored pole ends. Where quite a conglomeration of tissue had been thinly spread upon the glass, these organisms are often seen end on, and then look like cocci; cocci of the same character, size, and coloring reaction are also to be seen scattered through the specimen, but as the first described object is always to be seen more or less frequently represented, and as every competent patho-bacteriologist knows or should know that cocci represent a distinct phaze in the development of this class of micro-etiological organisms, I feel justified in asserting that the specimen before me is infested by but one species, and that its members belong to the ovoid-belted group of germs, which statement is also confirmed by the microscopic examination of properly-stained sections of the organs above described.

Liver.—The above description will answer equally well as to the manner in which the germs present themselves to the eye. They are very small, and appear even smaller in the section than when shown in smear-preparation, which is but natural, unless we accidentally come across an individual presenting its horizontal diameter exactly to the eye of the observer, and so situated in the upper portion of the thinnest of sections as to be in direct apposition with its covering-glass. In such cases not only the peripheral outline, but the distinctly colored pole ends and the equally sharply-outlined clear center, can be distinctly seen. As said, these objects cannot be distinguished from cocci when they present themselves end-on. They are sometimes to be seen in groups, and sometimes in chains of several members, but I have not seen them forming long-connected threads, as in the case of the swine-plague germs, especially in the kidneys. They are not only situated between the parenchymatous cells, but often in the bodies of the same; more especially are they represented in the smaller round cells present. No one but the merest tyro in the patho-biological examination of microscopic sections, no one competently versed in the technique of exact research, can or could possibly overlook these organisms, or mistake them for karyokinetic figures or the peculiar phenomena presented by mast-cells, both of which fail the clear outline of those organisms, and in general having more affinity for the coloring substance.

They can be distinctly seen in the capillaries, sometimes almost fill-

ing the finest for a short distance, and then again extending along their course as single individuals, which conditions are only to be seen in fortunately-made sections. In the larger vessels when filled with coagulated blood, and a thin section of the coagulum has been fortunately made, they will be found more plentifully represented than in any other part of the section. In the preparation of sections by the Carbol-Tuchsin method, great delicacy of technique is required, as there is extreme danger of the formation of a very delicate precipitate which the uninitiated might easily mistake for micro-organisms, but which can be thoroughly removed by the lege-artistic treatment of the section before mounting. In the kidneys of the same individual the same micro-organism is also profusely represented under the same conditions as those already described in connection with the liver. They were also present in the lymph-gland of the abdominal cavity.

"CASE 2.—Edwin Sing, Norwegian; died at Hospital Garcíní, October 24, 1888, at 2:30 P.M. Autopsy at 4 P.M."

Liver.—Interstitial tissue diffusely increased, with marked sclerosis in those places where the larger vessels and gall ducts take their course. While in some places it is markedly cellular, in others it is quite well organized, cellular structure being more or less wanting. In many parts the acini are distinctly outlined from each other by the presence of newly-developed connective tissue, in which may be seen pieces of the ramifications of the neoplastic arterial capillaries, the walls of the larger arteries are much thickened; in many places the walls of the gall ducts are so thickened that no lumen is to be seen. The parenchymatous cells are moderately swollen, and what is most striking is the entire absence of sharply-coloring nuclei in the greater number. In others the nucleus is colored indistinctly, while in others again it cannot be seen at all. These latter cells are completely filled with a granular substance, some of which is of a dirty yellow color, and but slightly refracting, being very fine. Intermixed with this substance is a more-coarsely granulous material, the individual granules of which possess a very strong degree of refraction.

Kidneys.—The general character of the microscopic lesions is the same as in the previous case; complications of the stroma of the organ being slightly more pregnant, and the sclerotic condition of the walls of the large arteries more marked. The engorgement of the sub-capsular blood vessels and those in the substance of the

cortex is greater, and the extravasation more frequentand extensive. The parenchymatous changes in the tubes are not, in general, so marked, casts being wanting. Cover-glass specimens made from these organs in the same manner as before, indicated exactly the same conditions as to the micro-organism present, except their more plentiful representation. Sections of the organs correspond thereto, the previously-mentioned pigment in the cells of the liver seriously interfering with the view in many places, however, but in thin spots, more or less clear from the same, the germ may be distinctly seen. In the kidneys they are most profusely represented, especially in the inter-tubular tissue and spaces, and are to be seen with great distinctness, the sections being most remarkably thin, as well as successfully colored. The variation in the extent of the uncolored belt which occurs during the process of evolution, and hence causes a variation in the length of these organisms, is to be distinctly seen. The coagulum in some of the larger vessels is almost completely filled with them. *No other organism is to be seen.*

"CASE 3.—William Nelson, German; died at Hospital Garcíní, October 15, 1888, at 7 P.M., of yellow fever. Autopsy at 7:30 P.M. the same day."

Liver.— Presents conditions exactly similar to those in the previous case, with the exception of a marked engorgement of the arterial circulation, as well as distention of the veins in places, and more complete sclerosis of the interstitial tissue surrounding the larger vessels, and less marked complication of the inter-acinous tissue. The *kidneys*, on the contrary, present similar parenchymatous changes to those described in the previous case, except in a more extreme degree, the granular character of the mass filling the tube being excessively marked. Cellular structure was almost entirely absent, as well as the presence of nuclei. The most marked change is a diffuse complication of nearly all the capillary structure of the cortex, the walls of the arterioles being so replete in nuclei as to make them almost as distinct as if injected. Accumulations of mono and multo-nucleated round cells are present at many points of bifurcation of the arterioles, and especially in the vicinity of Bowman's capsule. The cortical portion is marked by a few engorged vessels and an occasional extravasation. In the medullary portion of the section at hand is a large vessel with a ruptured wall, surrounded by an extensive mass of extravasated blood.

Examination for germs.—Smear-preparations of the above organs are especially characterized by their purity of representation, the absence of the coccoid form, and remarkable clearness in which the belted structure and polar characteristics are presented. Examinations of sections confirm the previous statements.

"CASE 4.—Magin Marquez, Spaniard; died at Hospital Mercedes, July 8, 1888, at 1:30 o'clock P. M. Autopsy at 4:30 P. M. same day."

Liver.—Sections of this organ are marked by the extreme degree of sclerosis in Glisson's capsules at those points where the larger vessels are collected, with extreme thickening of the walls of the gall ducts and encroachment upon their lumen. The veins are generally full of blood; many are markedly distended, which condition does not extend to the arteries, as in the previous case. The complication of the inter-acinous tissue is greater and less cellular in character than in the previous cases. There is also a diffuse complication of the intra-acinous connective tissue throughout the entire organ, with distinct encroachment upon the parenchyma, which, while less in quantity, has not undergone the same degree of acute degeneration as in the previous case.

Kidneys.—The conditions of the kidneys correspond more or less to those of the liver, those of a parenchymatous nature being of a minor degree, while those upon the stroma are extreme, especially those of the vascular system, which are even more marked than in the previous case. The Bowman's capsule seems to be thickened, and in some places the entire parenchyma has disappeared, its place being taken by granulation tissue. The cells bordering on the membrana propria are in general marked by clearly distinct nuclei, though an occasional one is wanting here and there. The balance of the contents of the tube is generally of a granulous character, though here and there a nucleus can be seen, and occasional hyaline casts also. Hemorrhages wanting.

Examination for micro-organisms of above organs.—Both smear-preparations and sections demonstrate the presence of the same micro-organism as heretofore described, with all the distinctness necessary.

"CASE 5.—Soldier; died of yellow fever, in military hospital, Havana, June 3, 1888. Autopsy five hours after death."

Liver.—Corresponds to that of the previous case, but has a more

marked sclerosis of the interstitial tissue in the great-vascular spaces, with very distinct thickening of the walls of the arteries, and almost complete occlusion of the gall ducts. The inter-acinous complications are not, however, as distinctly marked, while the diffuse intra-acinous complication of the stroma is about the same.

Kidneys.—Diffuse complication of the stroma; a very slight degree of parenchymatous degeneration.

Examination of the tissue of above organs for germs—confirm previous statements in *optima forma*, both in smear-preparation and section.

"CASE 6.—Soldier; died of yellow fever, June 6, 1888. Autopsy three hours after death."

Liver.—Interstitial complications less marked than in the previous case, being in general diffuse, without centralization at any particular point.

Kidneys.—Present the phenomenon of acute parenchymatous inflammation, with no complication of the stroma worthy of mention.

Microscopical examination of above tissue for bacterial organisms is but a simple confirmation of the results previously quoted, with this advantage, however, that the numerical representation of the germs is much more profuse than in either of the previous cases.

A very large number of sections, and an equally large number of smear-covering-glass preparations, were made from this material, *and it so happens that in but one of all these specimens* (most rigidly examined, not only once, but many times, and again most critically re-examined when the previous notes were dictated) *was there any other form of micro-organismal life present than that seen by Lacerda and Babes as early as 1884, and by me again in 1887, in some of the same material, and also by Sternberg.* This pollution consisted of but two examples of a slim-rod, with rounded ends, coloring diffusely and intensely, and having about the dimensions of the tubercle bacillus. Whether or not this is the bacillus of Gibier, I cannot say, as in none of the literature at my command is there a description of that organism sufficiently detailed to warrant the forming of even an approximate conclusion.

The other organism was present in every case, sometimes much more frequently represented, but failing in none. In the Lacerda material it was, however, most profusely represented, as has been previously mentioned.

It will be remembered that both Sternberg and Gibier more or less positively deny the constant existence of any organism in the tissues, though Sternberg admits this one distinctly, and infers others, while Gibier as distinctly says, "the blood, the liver, the spleen, and the kidneys, have been found free from microbes."

With even more dogmatic positiveness; aye! with the effrontery of absolutism, I assert that this organism is invariably present in all the tissues examined by me, and that he who cannot demonstrate it is deficient in microscopic technique. I freely admit that it is difficult to demonstrate when very sparsely represented, and only by isolated individuals scattered here and there among the cells; but if a small vessel filled with coagulated blood is cut appropriately, there can be no possible excuse for not seeing them. Perhaps it may be that my more extensive experience with this class of germs, and intimate acquaintance with their appearance in tissues, may be of value. But be that as it may, again I repeat, *that not only in the Lacerda material, but in a very large number of sections from these six Burgess cases, this one organism is present, sometimes and often seen end on, then looking like a coccus; sometimes lying full to the eye, and then presenting its two pole ends and clear middle piece, the latter varying much in extent in different individuals; sometimes seen obliquely, and again, if on the surface of the tissues and intensely exposed to tinction, sometimes colored almost diffusely; but no matter how seen, it is one and the same organism.*

I do not, however, rely upon the examination of sections to make such a diagnosis. One is liable to be deceived if he does that. Hence long ago I bethought me of some method by which we could handle alcohol-preserved material as easily and as surely as we can that absolutely fresh. After some experimenting I dropped upon the following plan, which can be promised to give as reliable results as those obtained from material taken immediately from a freshly-killed animal. Naturally, the result will also depend upon the freshness of the material when placed in the alcohol. In order to test its accuracy, I have repeatedly killed an animal purposely inoculated with a known germ, and have at once put pieces of the organs in alcohol, while at the same time I made fresh smears from the blood and organs, preserving the slides. At a later date I have then treated the alcoholic material in the manner to be described, and arrived at equally satisfactory results. It is scarcely necessary for me to say that this method

is fully as reliable as the use of fresh material; in fact, when the precautions are duly observed, it may be more so; for, if the material is properly removed, the piece washed externally in cor. sub. solution and then in distilled water, and from these into absolute (or 95 per cent) alcohol, the addition of any new organisms to the piece other than those present in the animal, is next to an impossibility.

THE METHOD.

The trouble with alcohol-hardened material has always been the uncertainty of what we saw, even in the thinnest sections, in the case of such very minute organisms as those of the ovoid-belted septicæmia producing group.

To avoid this: Take a perfectly clean, cotton-plugged, absolutely sterilized test tube, (or the requisite number.) Filter into it about the same quantity of a 1:10,000 caustic potash solution as you would use of gelatine or any other cultivating material. Sterilize this by heat. Let it stand then until assured it is negative. (When one is working much, numbers of these tubes may be prepared at once and kept on hand.) With sterilized forceps, lift the piece of organ into the neck of its bottle, and from the center, by other forceps, remove two or three small fragments and quickly place them in the potash solution leaving them there for some hours, or until the alcohol is displaced. Then on carefully cleaned and sterilized glasses, (if one thinks he needs so much care, and in such cases as this it is unquestionably necessary,) rub up these fragments, or such parts as are necessary to give a coating, as with fresh material; dry in air; flame, color, and mount.

Some of these objects color well, in sections, in Loeffler's Methylenblau caustic potash (1:10,000) solution, but when very sparsely represented, or when the sections are the least bit thick, this method will leave one in stick with regard to yellow fever material.

The only reliable and if properly used invariably successful method by which these organisms can be made visible, even in quite thick sections, and when only few are present, is by using the well-known Carbol-Fuchsin solution, so commonly used in "hunting" the tubercle bacillus; and here, too, we use heat, but very much more than is necessary to fix the color in B. tuberculosis. But:

First.—Wash the sections to be colored for a few moments in some absolutely germ-free caustic-potash solution, (1:10,000.)

Second.—Place them in a salt-cellar containing two-thirds caustic-potash solution, (1:10,000,) and one-third Carbol-Fuchsin (five per cent carbol acid in distilled water, 100 parts; alcohol, [95 per cent,] 10 parts; Fuchsin, 1 part. Filter and also filter each time on using, and place over slow flame until vapors rise and one-half of the coloring material has been evaporated. The slower the evaporation the better.

Third.—Wash in one per cent acetic acid, *in alcohol.*

Fourth.—Wash in distilled water to remove surplus and any sediment. Experience can only show how much color to remove.

Fifth.—Wash for five minutes in Ol. terebinth, two-thirds; alcohol, one-third.

Sixth.—Wash for five minutes in pure Ol. terebinth.

Seventh.—Wash for five minutes in xylol.

Eighth.—Wash in oil of cloves and mount in balsam.

Notwithstanding every endeavor upon my part, it was absolutely impossible for me to procure any material of any kind whatsoever from the various outbreaks of the yellow fever in our Southern States during the summer and fall of 1888.

Monopoly in original research on questions pertaining to the public health or national economy seems to be a new phase of the high protective policy in this tax-beridden country. However, another was more fortunate, and his wonderful results have been heralded to the world in the following resonant phrases:

DR. REEVES' DISCOVERY.

"Dr. Sternberg, of Baltimore, Md.," says the Chattanooga *Times*, "who has for the last six or eight years been investigating the yellow fever, with a view to discovering the specific cause, it will be remembered spent a portion of the summer at Decatur investigating the disease.

"While in Decatur conducting his investigations, he kindly sent to Dr. James E. Reeves, of this city, pieces from the liver and kidneys of several cases of yellow fever for microscopic examination with a view of searching for the specific germ of that disease.

"Dr. Reeves took these tissues and began his investigations. From a mounting of the very first cases, he found a micro-organism in them which led him strongly to believe that he had found the germ. He continued his research and thoroughly examined all of the tissues sent him from Decatur, and he found the same thing in all, more or less developed.

"He forwarded mounted slides to various microscopists and scientific men throughout the country, who one and all say that the result is exceedingly interesting, if nothing else, some declaring that it was the yellow fever germ. Mounted slides of these tissues were sent to Prof. H. J. Detmers, of the Ohio State University. These slides were photographed and forwarded to the Johns Hopkins University. The micro-organism is a bacillus, rod-like in shape, and is found in schools of millions in the tissues. So very much struck are the scientific men with the discovery, that Dr. Reeves has been invited to come to Johns Hopkins University, under pay, to make a demonstration of what he has found and the method employed. In accordance with this invitation, Dr. Reeves will leave for Baltimore the latter part of the first week of the new year.

"A *Times* reporter called to see Dr. Reeves, and asked him with reference to his discovery. The doctor was not prepared to say definitely what his find amounted to, but said:

"'The discovery of these bacilli in the tissues leads me to hope that I have found the germ of yellow fever, but as a matter of course we cannot say definitely until we have further carried on investigation. I can say this much, that in all my experience with micro-organisms I have never seen anything like that which I have discovered in the tissues furnished me from yellow fever patients who died at Decatur. I have examined these tissues in every way, and under different staining agents, and have always met with the same result, which leads me to believe that whether these bacilli are the specific germs of yellow fever, they are certainly an organism pertaining to and only found in the tissues of persons suffering from yellow fever. I shall go to Baltimore and consult with scientific men of Johns Hopkins University, and my experiments will be carried out, and we may then be able to tell what it is I have found; but until these precautions have been taken by making experiments as being peculiar to yellow fever cases, there is, of course, more or less uncertainty about it.'

"The reporter was shown the mountings the doctor had made from the various tissues, and saw a most interesting sight. The tissues were filled with red-like spots, hundreds of which might be put upon a blunt pin point."

It will be seen that Dr. Reeves said:

"The discovery of these bacilli in the tissues leads me to hope that I have found the germ of yellow fever. * * * I can say this much, that in all my experience with micro-organisms, I have never seen anything like that which I have discovered in the tissues furnished me from yellow fever patients who died at Decatur."

It will be seen that Dr. Reeves speaks of "yellow fever patients," using the plural number; but, what is of more value, he says: "*I have*

examined these tissues in every way and under different staining agents, and have always met with the same result." No one will more cheerfully admit than I that as a mere technician and manipulator of microscopic sections, Dr. Reeves has but few equals and no superiors in this country, if in any. I am no such delicate worker, for time is too precious with me to give any pains to appearances. Here it is results only, no matter how one gets at them, and with a very sincere disrespect for rule-of-three methods.

Naturally, on seeing such wonderful reports from Dr. Reeves, I was very anxious to acquire one of his specimens, and was fortunate enough to obtain one selected by himself as especially suitable to photographing.

This slide is distinguished by a label on each end — not the plain labels of ordinary workers, but the more elaborate affairs emanating principally from manufacturers and dealers in model slides. Upon one of them may be read "Dr. Jas. D. Reeves's Microscopical Laboratory, Chattanooga, Tenn.," and upon the other, "Yellow Fever Liver $\frac{5}{5}\frac{6}{2}$'."

When I received this slide it was colored in "Bismarck brown," and as the liver cells were very full of a glancing granulous detritus, it was only with the utmost difficulty that one could isolate the much more minute germs of yellow fever; but by careful focusing they could be occasionally picked out between the cells. I am writing on April 24th, but received this slide January 2, 1889. From that time until April 20th, I preserved it as it originally came to me, in order to show it to visitors, that they might compare it with my own, made from the various materials herein mentioned. No one has had any difficulty in seeing that apparently the same organism was in all of them. Desiring to have a better idea of the actual numerical representation present, I most carefully removed the cover glass and specimen and dissolved all the balsam, and re-colored it according to the Fuchsin method previously given. It can be truly said, and actually demonstrated, that the number of ovoid-belted germs present is greater in this specimen than in either of the others examined, and that by this method they can be so distinctly seen as not to allow of the possibility of their being mistaken for detritus granules, "mast-zellen," or anything else.

Let us now review this evidence a little. There seems to be no reason to doubt the genuineness of the material. Of that from Brazil, Lacerda has told us that:

"In all these preparations, (six persons,)* without exception, *I have found one bacterium exactly identical with that which Babes has already found in material sent him from here by me.*"

Now Babes described and illustrated that organism so well that when we are assured likewise that the material sent me from Havana by so accomplished a practitioner (and one holding such an important position as "Inspector of the United States Marine Hospital Service") as Dr. Burgess is all from "undoubted cases of yellow fever," it would be carrying skepticism to the verge of imbecility to doubt that it is one and the same organism.

To be sure, Babes failed in finding it in some other material, examined later, and Sternberg only found it in "two out of nine cases," of which he brought material from Brazil, perhaps the very same; and Gibier did not succeed in finding it at all; but on the other hand, as said above, Lacerda reports finding it "in all preparations without exception," and here we have the same organism also "*in all preparations without exception,*" *from "six undoubted cases" from Havana, and again, reported by Dr. Reeves,* whose accuracy of observation and delicacy of technique I personally know can be depended upon, who says of it: "*I have examined these tissues from yellow fever patients in every way, and with different staining agents, and have always met with the same result.*"

Although we do not know just how many persons the material examined by Dr. Reeves represented, we do know that in all these tissues no one has spoken of the presence of any other organism, and any one with a grain of pathological acumen must say, *that under such circumstances the organism present must have been the cause of the disease with which the individuals were diseased from whom the material in question was taken.*

What, then, was that disease?

Has not every observer who collected this material, even to Dr. Sternberg, asserted, without equivocation, *that it was all from "undoubted cases of the yellow fever"?* This being so, *then I unhesitatingly pronounce the germ present in all these cases to be unquestionably the etiological moment in the yellow fever, and that failure to demonstrate its presence in all such cases, as well as by cultures, by those having opportunity, is due to want of technical ability in most cases, though accidental failures must be allowed for in a few.*

*Virchow and Hirsh, Jahresbericht, 1887.

I am perfectly well aware of the extreme relish with which my estimable confreres in the world would point out any serious error in the work of so severe a polemist as myself; but I am equally well aware of the character of the few abilities I may possess, and of the painful exactness used when it is absolutely necessary. Some may think me overbold; but where caution is warranted by the evidence, I can use as much of it as any one, as will be seen in my treatment of some diseases later on in this report. What scientific investigators are seeking — must seek — are the exact facts, and no matter who makes an error, the more severe the exposure of the same the better for the world at large; and I certainly not only do not fear, but have so acted in my course as an investigator as to court all the criticism that can possibly be thrown against my work. Thus far the errors exposed have been made manifest by their extreme rarity; in fact, so rare have they been that not one error of fact has thus far been found in my published work, except one of misconception, which I myself exposed in a late publication.

Let us now return to Sternberg's address for a moment.

It has been noted already that he said: "In my researches by the method of staining thin sections of tissues hardened in alcohol, I have encountered several different micro-organisms, but no one of these has been found in a series of cases," (l. c.)

Here we have, however, one and the same organism, not only found in "a series of cases," but also the only one found by other observers with any degree of constancy, and Lacerda has also told us that he found it "without exception" in six cases of the disease.

This is positive evidence, and every competent investigator knows the value to be placed upon negative evidence in such cases. I am perfectly aware of the nature of the rules promulgated by Robert Koch as necessary to be fulfilled before the complete chain of scientific evidence can be said to be perfect. They are as follows:

First.—One organism must be found in an absolutely pure condition in the tissues of an individual that has perished from a given disease.

Second.—That micro-organism must be isolated from those tissues and cultivated in an artificial manner independent of them.

Third.—The same disease must be produced in healthy individuals of that species of animal life in which such a disease occurs under con-

ditions of natural infection, by the intentional introduction of such organisms either by a natural way or artificially.

Naturally, the last postulate is very difficult to carry out in relation to diseases limited in their natural occurrence to the human family.

Undoubtedly the above rules are correct; but when we cannot follow them out, as in many human diseases, what then? Are we to neglect the teachings of common sense?

Has Koch fulfilled them in Asiatic cholera? And yet no competent person doubts the etiological importance of the celebrated Komma in that disease.

Those are the rules of the extreme bacteriological school; but not one competent pathologist would be guilty of carrying them out to such a severe conclusion as Sternberg, when he says:

"Evidently, if any one of these micro-organisms was found in a considerable series of cases, the fact would be decidedly significant, and would afford presumptive evidence that the parasitic organism found bore some relation to the morbid process.

"*But even if one and the same micro-organism was found in every case, the final proof of its etiological import would depend upon its isolation in pure cultures, and the production of the characteristic phenomena of the disease in one of the lower animals, or, in the absence of a susceptible animal, in man himself.*" Whew!

That is what I call "straining at a gnat and swallowing a whole camel." Such language as that can only emanate from one most seriously wanting in clinical and necroscopical experience, but above all in pathological acumen and rational common sense. I am perfectly well aware that the whole ultra-bacteriological school will raise a hurricane of indignation about my ears, but nevertheless I do say that such language as that is absolutely insane under certain circumstances. It is on a par with some who teach that a physician cannot possibly diagnose tuberculosis except by the microscopic demonstration of the tubercle bacillus. What would Laennec have said to such a remark as that? What would Virchow say to a student out of his school who should present any such absurd doctrine?

We who are busy day in and year out in the examination of small vials, or specimens, brought by farmers from all over such states as Nebraska, know a thousand times better than to insist upon the necessity of any such nonsense being carried to that extreme.

Were that true, every time a farmer brings me blood and I find

Bacillus anthracis in it, I should have to wait to inoculate and cultivate before I could give him an answer or advise him what to do.

Were that really so, then all our clinical and pathological knowledge and experience would be of little avail.

Were that so, then the sacred few who have taken holy communion at the bacteriological table of Robert Koch are the only masters and leaders, and all others who have gone before and all of us unsanctified ones who have not tasted of that special fruit of the tree of knowledge, are ignoramuses, and unfit to be trusted.

But let us come to the point.

What is the yellow fever as a pathological entity?

What is its nature as a whole?

Leaving out of consideration Drs. Sternberg and Gibier, all pathologically-competent observers, past and present, *agree that the yellow fever is a septicæmia.* Being then, a blood poison, the germ must be in the blood — is in the blood — as my sections show. Not only that, but we have mountains of exact testimony showing that other microorganisms of this group do produce septicæmiæ, and a specific septicæmia, in certain well known species of animals. For instance, the southern cattle plague, the swine plague, hen cholera, the German Wildseuche, and my new corn-stalk disease, are all septicæmiæ and all caused by an ovoid-belted germ. But each of these diseases has something peculiar about it which makes it different from the others. The Wildseuche attacks deer, cattle, and swine; the swine plague, hogs only; hen cholera, some members of the feathered tribes; the southern cattle plague, cattle only; while the corn-stalk disease is only known, to me, to attack cattle. It certainly does not hogs, but may other herbivora. Now, *given an outbreak well marked clinically, and in which we find the well-known necroscopical lesions, in a blood disease of this character, if a competent microscopist finds in the blood and tissues of an animal killed, or which died and was opened in due time, one form of microorganismal life, and no other, or even if in smear-preparations he does occasionally find some extraneous thing, I say that he does not know his business if he is not absolutely certain that that organism is the cause of that disease; experiments or not, cultures or not.*

For instance: Our farmers do not need to be told when the southern cattle plague is among their cattle. The first thing they at once discover is that Texas cattle have been imported, and have wandered

or been driven about. That fact known, the season summer, cattle dying in numbers with red urine, constipation, and high fever, no other means are necessary to make a diagnosis. These factors being present, we open such an animal, having killed it purposely, and under all precautions take away blood, pieces of organs, etc.; and on making a microscopic examination of that material we find but one organism present, and that in great numbers: is there any actual need of experimentation to complete the diagnostic evidence?

To my mind not!

In the next article will be found the details of the discovery of an organism without any knowledge of either of the necessary factors. Did I have any doubts that that organism with which the blood was densely charged was the cause of the disease which killed those animals, though I had no data by which to name it?

I trow not? Had I had such want of faith in my own judgment—had I so lacked in common sense—I should be unworthy of the position I occupy or reputation I enjoy. Taking then the reliability of the diagnosis into consideration, knowing the nature of the disease, and with perfect faith in the ability of these observers, who all say the material I examined was from "undoubted cases of yellow fever," I am willing to risk some considerable reputation that the germ herein described as present in all this material is the specific cause of yellow fever, as well as that the description of the morpho-biological phenomena presented by the germ of the southern cattle plague will largely be found applicable to this.

THE SWINE PLAGUE AND SOUTHERN CATTLE PLAGUE GERMS DIFFERENTIATE THEMSELVES VERY SHARPLY BY THEIR APPEARANCE WHEN CULTIVATED UPON POTATOES.*

If we properly prepare (see text-books) and sterilize some nice, clean potatoes, and then place them (lege-artis) in a sterilized, moist cultivating chamber, and inoculate the cut surface of some of the potatoes from pure agar-agar, bouillon, or other cultivations of the microorganisms of these two diseases, we shall invariably find that they can be readily differentiated from one another in the course of from twenty-four to forty-eight hours after the surface of the potato has been inoculated.

*See the next bulletin in this Report for other points of value.

The growth of the germs of the American swine plague will invariably present a peculiar yellowish-gray shade to the eye.*

On the other hand, the micro-etiological moment of the southern cattle plague will, with equal constancy, present a growth of the most delicate straw color during the first day or two of its development, but which soon begins to show a delicate pinkish-red-yellow, and finally quite a decided brick-red-yellow shade, as the cultivation becomes antiquated; this reddish shade begins and grows most intense at the center of the growth, leaving it more yellow toward its peripheries.

THE DEPORTMENT OF THE GERMS OF SWINE PLAGUE AND SOUTHERN CATTLE PLAGUE IN BEEF-INFUSION GELATINE.

As what is known to us as beef-infusion gelatine cannot be used in hot weather, or when the prevailing temperature is above 75° F., (23° C.,) on account of its becoming fluid, I could not use this material until the last moment, and only prepared the first of the season on October 1st, and on the 2d was enabled to inoculate tubes of this material from pure cultivations of the germs of southern cattle plague and hog cholera. This beef-infusion gelatine is an invaluable medium in the technique of bacteriology, for two essential reasons: First, being transparent one can see what is going on in it; and secondly, many micro-organisms cause the solid material to become fluid, or present peculiar phenomena to the eye, while others do not cause any change in it, but may grow in a peculiar manner.

Now these germs belong to the latter class, as do also those of hen cholera, and the peculiar disease known as "Wild-seuche" in Germany, which affects the deer tribe and cattle and hogs, and belongs to the same blood-poisoning group of organisms. When we inoculate tubes containing this beef-infusion gelatine from the pure agar-agar cultures of these organisms, we shall observe that the germs do not cause the gelatine to become fluid, and that it never becomes so, so far as any influence of the germs goes, if the culture from which the material had been taken was a pure one—that is, contained no other form of micro-organismal life than the germs in question. The germ of swine plague, however, has its peculiarities: it slowly spreads over the surface of the gelatine as a delicate cuticle; but

*See plate II.

as these cultures are made by puncturing the gelatine with a wire, the germs are carried into that substance by the wire. Here we observe that wherever the wire has left a germ in its passage through the gelatine, a small colony develops, giving to the puncture the appearance of a delicate thread, with knots along its course. In the end, these colonies unite, and give the puncture a ragged-edged appearance along its entire length. As the germs of the German swine plague and rabbit septicæmia and the "Wild-seuche" all do the same thing, Hueppe has asserted them to be the same organism, and therefore that all these diseases are one and the same; a view I can not agree with.

THE CHIEF MEDIUM OF THE INFECTION OF OUR NORTHERN PASTURES AND THE EXTENSION OF THE DISEASE IN THE SOUTH IS THE MANURE OF THE INFECTED ANIMALS.

Having described the germ, and how it was found, and proven it to be the cause of the southern cattle plague, it is now necessary to prove its existence in the manure before we discuss the part that material plays in infecting the land and extending this disease. In regard to the manure, a very surprising yet confirmatory discovery has been made.

First.—How do we know the germ is in the manure?

The fæces of all animals contain a great many varieties of micro-organismal life.

We have now in the laboratory some old dry manure that came from the Texas-infested pasture at Tekama the middle of July last, (1887,) and which has been kept in an air-tight specimen glass ever since.*

We have also a similar glass which was previously sterilized, into which the manure of the S. and G. steer (upon which we made the autopsy recorded in another part of this report) was carefully emptied from the large intestine. The latter is fluid, while the first is more or less dried up, but still contains sufficient moisture to preserve germ life Now if an artificial culture is made from the old manure that came from the Texas cattle April last, (1887,) in sterilized bouillon, a very surprising result will follow. There are only three varieties of

*That was written in March, 1888. In March, 1889, I tested the same manure upon mice, and found that the germ still retained its virulence. Naturally, it had been at a temperature of about 20° C. all this time.

germs in it, and one exactly resembling the one we have proven to be the cause of the disease will be found to predominate. And how can we tell, to a reasonable degree of certainty, that it is the germ?

It can be isolated and cultivated in the ways known to those who pursue this kind of research. It will there be found to grow exactly as the other did, even on potatoes, and it will kill ground squirrels and mice. The cattle at our command have been inoculated with pure cultures directly from animals, and it will not affect them. Whether such a result has any practical result or not we cannot now tell. We may eventually find out.

Now while this manure is replete in these micro-organisms, a microscopic examination of the fresh manure, directly from the intestines, shows not only a greater variety of germs, but, while present, this one variety is not nearly so plentiful as in the old dry manure. Inoculations from a diluted fluid made of fresh manure also prove fatal to ground squirrels.

Now these two germs being so far alike and conforming in every particular in their development in and upon artificial media, what do these facts teach? The results exactly correspond to our knowledge of the natural biology of this micro-organism.

The animal organism is not its natural home. The earth is. Now the old manure, by processes of evaporation, absorption, and decay, had gradually become more and more earthy and lost its animal fluids and perhaps some chemical components. It had thus become more like the natural soil, and the temperature at the time having been for a sufficient period similar to that under which the germs thrive in Texas, a rapid proliferation had taken place in the unbroken pad of manure, for this collection was made of still unbroken pads which laid in the same place where they were originally dropped by the Texans. Now I assume, whether right or not the future must show, that the reason that the specific germs are fewer in the fresh manure directly from the sick animal is, that it does not offer quite so favorable conditions for their rapid development as that deposited on the field at a favorable time of the year.

This is a supposition which we will endeavor to prove by mixing that manure with some sterilized earth in a sterilized vessel and exposing it constantly to the same temperature that prevails in summer, that is about 90° F.

THIS HAS BEEN DONE AND THE HYPOTHESIS SHOWN TO BE A FACT.

Some fresh earth was placed in a tin pan and exposed to a temperature of 250° C., in a dry, hot air, and sterilized for two hours a day for seven days. To it was then added some freshly-distilled water, which had been boiled for fifteen minutes a day for three consecutive days. This moist, earthy material was then carefully mixed with equal parts of the manure from the S. and G. steer, and placed in a moist chamber in a thermostat at 90° C. for five days, when bouillon cultivations were made from the same, and examined microscopically at the end of twenty-four hours.

Instead of the mixture of micro-organisms found in the manure when freshly taken from the intestines of the diseased steer, this bouillon culture from the earthy material that had been exposed to summer heat contained almost exclusively a germ exactly corresponding, morphologically, with that of the southern cattle plague previously described, and but a very few long and short rods of other species. The former were then isolated by plate cultures and then inoculated upon potatoes and in gelatine. The results corresponded exactly with every necessary condition.

Ground squirrels and mice were next inoculated with but one drop each on the inside of the thigh. Mice died in twenty-four hours; ground squirrels in thirty-six and forty-eight, respectively. These experimental results fully confirmed my theoretical conclusion that if this fresh manure was rendered earthy and exposed to a high summer temperature the germs would rapidly develop in it and gain in virulence.

PRACTICAL EVIDENCE THAT THE MANURE MUST BE THE CHIEF MEDIUM BY WHICH INFECTION OF THE LAND OCCURS.

The Texas cattle arrived at Tekamah April 1, 1887, and it froze hard for about two weeks subsequently. July 1st the southern cattle plague broke out, or after a lapse of ninety days. Now had the germs been bound on the saliva dropped by the Texans and not destroyed by the frosts, the disease must have broken out in our native cattle much earlier, for it only takes twenty days to cause it after infection has taken place, but it did not appear until July 1st.

Now where are these germs all this time?

Locked up in the manure!

So long as the pad dropped by infected cattle (Texas or native) remains moist, soft, and cohesive, there is no danger to native cattle placed upon the pastures where such cattle have been.

The results at Tekamah prove that to be a fact. The second outbreak demonstrates it even more than the first; for the climatic conditions were favorable all the time between the first and second outbreak, yet there are only twenty days actual variation in the time in which the eruption took place if we carry the first outbreak to a period when climatic conditions made infection possible.

The very freezing of the surface of the pads of manure helped to put them in a condition favorable to the preservation of the life of the germs within. It made a hard close crust on the outside of the pad of manure, which was also increased by the evaporation caused by the ever increasing heat of the sun in the day-time. These two factors together also helped keep and increase the heat of the inside of the pad, which gradually increased, thus favoring the proliferation of the germs enclosed in the pads. This, in connection with reasons given above, accounts to my mind for the greater number of these germs in the old pads of manure than in the fresh manure from the intestines of the diseased steer mentioned previously. These old pads must then have been replete in germs and well prepared to cause a rapid infection of the pastures when the temperature and elements together had so acted as to dry them up; which, with the feet of the native cattle, also helped break them up and distribute this material over the pastures, where the germs found a suitable place to live and conditions favorable to their development in the earth at the roots of the grasses at the prevailing temperature.

After the pasture had thus been prepared, the natives really become exposed to infection, and not before, no matter how long they may have been upon it previous to that time. Now in this second outbreak, that is, where S. and G.'s natives infected their own pasture, leading to the succeeding outbreak in the balance of the herd, the same cycle of events had to be passed through with. The twenty-three sick cattle themselves did no harm. They, as cattle, were not dangerous; had they been, the disease would be contagious. Their manure was the source of and cause of infection to the native cattle left in the pasture, not only when they escaped and were put into the Texas-infested pasture, but when they were returned, and re-

mained there after twenty-one of them were dead and buried. This manure was not dangerous to the balance of the herd so long as it remained moist and confined to its original pads; only when it became dried and broken up by the cattle's feet and the elements, and the germs distributed over the earth and protected by the roots of the grasses and proliferation had taken place, did the pasture again become dangerous. Here again a long time elapsed before actual infection took place, fixing the period at September 1st, or thereabouts, fifty-three days.

This time appears unusually long when we consider that the climatic conditions were far more favorable to the rapid infection of the pasture than in the first case where the Texans were the cause earlier in the season.

If we look carefully for the cause of this phenomenon we can easily find it in the Texans themselves. They came here with their intestines freshly loaded with the infectious principle taken directly from its native fields, where it develops in its primary and most active virulence, and in this condition the Texans planted it upon our soil under conditions more or less favorable to its retention of that virulence, at least much more favorable than when it had passed through one more generation of native cattle and been taken from pastures, under telluric and climatic conditions, which, as we know, were favorable to the development of the germ, but by no means its native heath. Now, in the second outbreak the germ had been taken by the second lot of natives from this foreign land, but it had passed through one lot of natives before it got there, and had lost somewhat in virulent activities thereby, as was shown by my experiments direct from the cattle at Roca, and especially with the fresh manure from the S. and G. steer, though one generation of artificial culture alone stood between before any cattle were inoculated, which may also have exerted some weakening influence. One thing is sure, though my cattle were sick and off their feed and shrank much, and had a temperature of 107°, 108°, 107° F., still they would not have died, hence I killed the sickest one when the temperature had fallen in twenty-four hours from 108° to 107° F., and found most conclusive evidence of the existence of the southern cattle plague.

SOUTHERN CATTLE PLAGUE PRODUCED BY INOCULATION WITH A PURE CULTIVATION OF ITS MICRO-ETIOLOGICAL ORGANISM.

On Saturday September 10, 1887, I inoculated a large red cow, which was very wild, and a five months' old black steer, with a pure bouillon cultivation obtained from the outbreak at Roca, Neb. It was impossible to examine the cow closely as she could not be caught except with difficulty, but she fell off in her feed and condition and became somewhat constipated, but recovered.

The black steer was off its feed, hair bristling, stood much by itself, respirations very much accelerated, but not labored, temperature elevated. Wednesday, the 14th (when first observed unwell), 4 P.M., 42.50° C.; Thursday, 15th, 4 P.M., 42.25° C.; Friday, 16th, 41.50° C., 9 A.M.

Seeing that this animal might recover, and desiring to see the effects of the inoculation, I killed it, and found the following conditions, which, when compared with the autopsy notes of the S. and G. steer, leave no doubt as to the nature of the disease, though the organs of the calf were not nearly so severely complicated as those of the steer.

NECROSCOPICAL NOTES OF BLACK STEER INOCULATED WITH THE GERMS OF SOUTHERN CATTLE PLAGUE.

Blood of the peculiar color and lac consistency which is more or less characteristic of the southern cattle plague. (Some freshly flowing blood was caught in a sterilized bottle as it spurted from a cut artery, and the characteristic micro-organism afterwards found in it.

Paniculus adiposus, somewhat atrophied and of a decidedly yellow color, costal peritoneum and omentum, as well as the serosa of the large intestines, were of the same color, interrupted by numerous petechial spots. The small intestine was of a general diffuse pink-red color variegated by engorged vessels and a few petechial hemorrhages. Blood vessels of mesenterium engorged; lymph glands swollen and œdematous; interstitial and sub-capsular vessels injected. Other lymph glands the same. Abdominal cavity contained about two quarts of a straw-colored fluid. Spleen swollen, full of blood, and somewhat soft; weight, 2¼ lbs. (Animal five months old.)

Liver swollen, edges rounded; cut surface of a yellowish gray-red color; opaque; acini swollen; central vessels invisible; inter-acinus filled with blood; each acinus was most beautifully demarcated by delicate lines of a bright yellow color, which represented the inter-acinus and distended gall ducts. Gall bladder distended and full of a dark greenish-yellow material; mucosa stained of the same color.

Kidneys swollen; the outside surface was most beautifully marked by the injected condition of the inter-tubular vessels, and presented as fine a picture of natural injection as could be desired, as well as illustrating the earliest stage of that general renal engorgement, which is represented by the intensely swollen, bluish-red, diffusely colored kidney, so often seen in this disease under conditions of natural infection. See Plate VII.

Stomachs more or less full.

Mucosa of the fourth stomach very much swollen and of a diffuse dark pink-red color, interrupted by numerous engorged vessels, ecchymoses and diffused hemorrhagic centers. Mucosa of duodenum and jejunum swollen and of a yellowish-red color; Peyer's plaques and solitary follicles much swollen; the large vessels engorged; occasional hemorrhages in the mucosa; contents semi-fluid. The yellow staining of the mucosa of the anterior part of the small intestine was lost in the ileum, though here the membrane was also swollen, but of a diffuse pink-red color, otherwise the same. Mucosa of the large intestine swollen, and of a delicate, diffuse pinkish-red, with some engorged vessels to be seen; this condition increased in intensity from the beginning of the rectum towards the anus, where the mucosa was intensely swollen, and of a dark pink-red color, with small hemorrhagic centers, especially upon the crests of the rugæ; contents more and more solid until it became quite hard in the posterior portion of the rectum.

Nothing abnormal in the thoracic cavity except that the lungs were slightly hyperæmic; the bronchial lymph glands swollen, red, and œdematous; the myocardium opaque, anæmic, yellowish grey-red in color, and somewhat soft. Mucosa of the trachea and bronchial tubes swollen, vessels injected, and a few petechial centers present.

Bladder half full of a straw-colored urine; vessels of the mucosa somewhat engorged.

Microscopic examinations — covering-glass specimens — of tissues

gave the characteristic bacteria. Cultures and subsequent inoculations of gophers gave the necessary positive results.

The original culture from the Roca cattle killed ground squirrels in an average of forty-eight hours, while the cultures from my inoculated steer were not certain on the same animals, killing some in three or four days while others recovered. I then went back to the original Roca material. It still had its original virulence in an inoculated gopher; it died, as usual. From the mashed-up liver of this animal in (distilled and scalded) water I then inoculated another ground squirrel. It took four days to die, and in inoculating another with material from the last, the germ had lost its fatal effects, though the animal was sick.

While we know that the bovine organism has in it some unknown quantity which renders it susceptible to southern cattle plague, we still know that it is not the natural habitat of that germ; hence, it is but reasonable to suppose that the germ must gradually weaken even in passing through cattle. It is much to be regretted that I could not use other animals except ground squirrels and mice, and test this germ on them in regard to this question; but until now I have never had a proper place to breed such animals, so that I only had on hand a very precious collection of rabbits* and Guinea pigs, which have been acquired for breeding purposes, to supply us with necessary material in the future.

Our experiments with squirrels, however, give experimental proof, which cannot be gainsaid, that the germ does lose in virulence in passing successively through several generations, and our cattle experiences at Tekamah show the same thing, which accounts for the slowness with which the natives caused the infection of their own pasture and the second outbreak in their own kind.

The manure pads had not only to have time to dry out and be dispersed over the field before the infection of the same could occur, but the germs also had to have time to recuperate, and acquire their natural energy in the soil even, before an active infection of the second lot of natives could take place.

These remarks will, I think, suffice for this occasion, and are sufficient to show not only how infection occurs through Texans, but also how it may, and has, occurred through the agency of infected natives.

* I have discovered that rabbits possess natural immunity toward this organism, at least to ½ ccm. injections.

By again referring to the table of outbreaks, it will be seen that these outbreaks at Tekamah are the only ones that occurred in the history of the disease where Texans came north early enough to allow time for the full cycle of events to occur necessary to the secondary extension of the disease through natives to natives.

Part III.—Nature of Southern Cattle Plague and the Yellow Fever.

Pathological Anatomy.—Prevention.

NATURE OF THE SOUTHERN CATTLE PLAGUE.

In the consideration of this question it will be impossible to avoid repeating somewhat that which has already been said in our consideration of the etiology of this disease, but in a disease of such vast importance such repetition is certainly pardonable.

The nature of any given disease is not the simple question that many might think, because diseases have different natures, according to the standpoint from which we approach the subject.

First, we have to consider a disease according to its origin, and the relation it bears between diseased and healthy individuals.

Second, according to the changes produced in the animal organism by the disease.

Let us consider these in their order.

The first question then for us to determine is, as to whether the southern cattle plague is an

ENDOGENOUS OR EXOGENOUS DISEASE.

What constitutes an endogenous disease?

An endogenous disease is one which finds its primary and only origin in some given animal species, and passes directly from one animal to another of the same species, or to healthy animals of that species, either by direct contact or from their coming in contact with some effluvia, secretion, or other material which has come from an already diseased animal. Examples: Syphilis, contagious pleuro-pneumonia, the rinderpest, and spotted typhus.

The above definition is, however, too narrow to include all the attributes of an endogenous disease.

While an endogenous disease invariably finds its primary origin in some one species of animal life, they are not all, like the above, limited to that one species in their action; there are some which, while having such a racial origin, still have the ability to infect other species of animal life than that in which they primarily occur, and in which they are generally found. Examples: Rabies, glanders, and the foot-and-mouth diseases, all of which are animal diseases, as well as the small-pox in man.

It is a singular fact that while most of the endogenous diseases of man are limited to the human species in their extension — syphilis, mumps, measles, scarlet — those of animals have a far more extended dispersion over the animal kingdom, inclusive of man, when accidental circumstances favor such extension.

At the present moment I can think of but one disease that is exogenous and still gives any just grounds for assuming that it has an endogenous character, and that is diphtheria; but even in that disease, it is my opinion that if we examine the question very closely we shall find that it is not endogenous, and that in every case accidental inoculation must take place in the healthy individual, and that, with due care, unless the surroundings are the center of its origin, the disease need not necessarily extend to healthy individuals per contact with sick ones.

How then does an exogenous differ from an endogenous disease?

In this way: *An exogenous disease is one which invariably finds its primary origin not in but outside of an animal organism. That is, in the earth, where its microbic cause develops under certain conditions of the climate and soil which offer favorable climatic and telluric influences to its development. Such diseases are always local in their origin. The earth bears the same relation to exogenous diseases that the animal organism does to endogenous; that is, they both form the primary center of development in their respective class; but with this difference: the focus of primary infection is fixed in exogenous diseases, while it is movable in endogenous.*

Much uncertainty and ambiguity has arisen in the minds of practitioners with regard to exogenous diseases, because, under favorable circumstances, infected individuals can become the means of their extension (indirectly) between diseased and healthy individuals.

Hence, without any mature reflection, such diseases have often been pronounced "contagious." In fact, there is far too much laxity in vogue, far too much ignorance of the philosophical use of medical technology among physicians upon this very important point, which has led to much unnecessary confusion in the minds of the laity, and renders it unnecessarily difficult to bring them to a clear understanding of suitable and applicable means of prevention.

As an example of this ambiguity, and, one might truly say, of logical ignorance of the use of medical language, we have only to refer to the reports of the United States Department of Agriculture upon the southern cattle plague.

Mr. Salmon says, of this southern or Texas cattle plague, that—

"There is no doubt that it is a difficult matter to understand how it is possible for the native cattle of a section permanently infected with a contagious plague to resist the influences of the contagion with which they are surrounded.

"It is equally difficult to understand how apparently healthy cattle can distribute this contagion for so long a time after they leave the infected district.

"It is not less difficult to understand why the cattle really sick of this contagious disease do not convey the contagion to others." Report 1883, p. 20.*

It is thus to be seen that Mr. Salmon has no definite idea of the real difference between an endogenous and an exogenous disease. The foregoing remarks sufficiently show that a proper differentiation between such diseases is not such a very "difficult" matter to any one having the requisite ability.

Above we have spoken of the part diseased individuals may play in the extension of exogenous diseases. Let us take anthrax, the most virulent of them all, for an example. If we have a case of anthrax among a stable of cattle in the winter, there is very little danger of any of the healthy ones becoming ill through that animal, if ordinary precautions are taken; but if the same thing should occur in the summer, when flies are abundant, there would be great danger of every animal present becoming infected. On account of this fact, many observers have unreflectingly said that anthrax is an endogenous disease.

* See also the latest report upon "Hog Cholera—1889," by the same author.

It has also been said that in endogenous diseases the infected animals were movable centers of infection; that is, wherever they go they carry with them danger of directly infecting other susceptible animals. Now something similar occurs in exogenous diseases. The infected animals may also become movable centers of infection; but in this they differ much from those diseased with an endogenous disease. They do not infect other animals directly, but they bear the same relation to the land that animals diseased with an endogenous disease do to healthy susceptible animals. They can and do infect new land, and may thus be the means of extending an exogenous disease over a broad extent of country, but only so long as they are actively diseased or have the inficiens within them, brought by them from its primary locality. This they do by means of their excretions. In this way anthrax, swine plague, southern cattle plague, the cholera, and yellow fever, originate and become extended from one place to another; but no matter how much they may become extended, they ever remain local diseases.

Now while individuals thus complicated by an exogenous disease may play an essential role in its extension, still there is this difference between the "seed," if I may be allowed the word, planted by them, and infected material dropped by and from an animal having an endogenous disease. In an exogenous disease the infecting material pollutes the earth, and finding there its natural nutriment, if the climatic conditions are not unfavorable, it will retain its virulent activities for an indefinite period, and in many cases acquire a degree of virulence a part of which had been lost in the animal organism; notwithstanding the severity with which it acted in the same, it goes on multiplying indefinitely. In an endogenous disease the circumstances are entirely different. Here, the animal organism forms the natural home of the inficiens, but when it is dropped outside of the animal organism, in some effluvia from it, it soon loses its virulent activity, and does not multiply in the same manner as the inficiens of an exogenous disease. Now any one can easily see that these are very essential points of difference, and that they distinguish these two classes of disease so sharply from one another that no one need be in danger of mistaking the one class for the other if they have sufficient education, observing powers, and logical acumen.

There is still another class of exogenous diseases which it is nec-

essary that we define, because they are very apt to be confounded with the previous one. These are known, or should be, as the

MALARIAL-INFECTIOUS.

How does this class differ from other exogenous diseases?

Both are local in their origin; but with this difference, the malarial-infectious diseases always remain local. In both classes a susceptible individual must be in, or upon, an infected locality in order to become infected; but while in non-malarial exogenous diseases a diseased individual can carry the inficiens, and thereby be the means of infecting other localities that may never have been pestilential, the case is quite otherwise in the *malarial-infectious, in which a diseased individual only becomes ill when living in such an infected locality, but has no power of infecting other localities, though he may leave the place where he became diseased and take up his residence in a place where it never has existed.* Example: Fever and ague. They are both local, land diseases, nevertheless. In my opinion, the air, as a disperser of infection only, plays a far more important role in the infection of individuals in malarial-exogenous than in the non-malarial diseases.

We have also said that the nature of any given disease may be, again, determined by the lesions, or disturbances, caused in the animal organism. This differentiation is known as the pathological anatomy of the disease. Taken together with the determination of its nature with relation to the disposition of a disease between sick and healthy individuals, we have what is known as the pathology of a disease. Pathological anatomy is purely a descriptive part of medical science; it describes the appearance of what has taken place in the diseased organism only. Pathology, on the contrary, is a reflective, theoretical science; it endeavors to explain how these changes have occurred; how the cause acts, and to differentiate between those lesions directly produced by the cause, and those which are secondary, or accidental, complications, which find their origin chiefly in the conditions directly excited by the first or primary cause of the disease.

Pathological anatomy can be acquired by any one who has good eyes and applies himself diligently. Pathologists, on the contrary, are born. They have been very rare. Good observers and recorders of natural phenomena have always been sufficiently abundant, but

correct interpreters of the same have been seldom. It is not alawys an easy matter to sift all the facts and determine the actual place of a disease according to the relation it bears between sick and healthy individuals; but difficult as that is, it seems to be still more so for observers to differentiate between the lesions produced by the specific cause directly and those of a secondary nature. We have been obliged to discuss this point very minutely in our article on swine plague.

To which, then, of these three great classes of pestilential diseases previously considered, does the southern cattle plague belong? THE SOUTHERN CATTLE PLAGUE IS A STRICTLY LOCAL EXOGENOUS DISEASE, and yellow fever is the same.

Having shown that the germ of the yellow fever in all probability belongs to the same group of ovoid belted organisms as that of the southern cattle plague, it now devolves upon us to support that assertion by calling attention to the very exact

RESEMBLANCES BETWEEN THE SOUTHERN CATTLE PLAGUE AND THE YELLOW FEVER.

In the preparation of my book upon "The Relation of Animal Diseases to the Public Health," while a student at Berlin, I was especially struck with this point, but did not then know enough of the bovine pestilence to more than call attention to it, as I did not have much American medical literature at my command. Since beginning—or better, completing—these researches, it has been necessary for me to review the subject, and I find that medical observers, especially those who have given some study to the southern cattle plague, have been struck with the same idea. Hence I cannot better open the subject than by a quotation from the most able of them all.

The best study of this bovine pest, in the past, has been that made by the metropolitan board of health, in New York City, 1867. In their report they make the following remarks:

"We have found a remarkable association of analogies to exist between the Texas cattle disease and the yellow fever as witnessed in the human family. It may be remarked that we feel fully warranted in adopting the expression used by Dr. Stiles, in his report, 'that the Texas cattle disease, when judged by its pathological lesions, might be termed the yellow fever in cattle.' A detailed account of these analogies need not be presented here, but it suffices to state that the

points in comparison in these two pestilences are so well marked as to warrant the belief that the actual demonstration of the precise nature, origin, propagation, and pathological effects of the infecting principle of either of these two pestilences would throw such a flood of light upon that of the other as to enable medical men soon to grasp and unfold the hitherto mysterious laws that govern the propagation of yellow fever. Let it be understood, however, that we do not presume that these two pestilences are identical! We simply assert that they are wonderfully analagous in essential and constant attributes in their pathology and in certain chief points, but not in all of the phenomena and habits of their respective principles or agents of infection." N. Y. State Trans., 1886–7, p. 1093.

The value of the scientific investigation of the infectious animal disesaes, as a contribution to comparative pathology, and as a means of throwing light on many human diseases of the same character, was so well appreciated by the metropolitan board of health of New York, in this report, that the writer may certainly be pardoned from quoting further, especially as it demonstrates the true value of the experimental laboratory which we have been so successful in establishing in Nebraska:

"Upon this subject the leading medical philosophers and hygienists of our day have for some years past been urging the necessity and duty of making careful and thorough scientific investigations in regard to the infectious maladies which afflict cattle, sheep, and other domestic animals. It requires no argument to show that both the labor and facilities, as well as the satisfaction, in making exact observations upon tame animals that are wholly under the control of the medical observer, and subject in all respects to his absolute authority, even to any kind of experimentation (experiments that are, of course, not cruel or barbarous), and subject, at the observer's arbitrary decision, to slaughter and to the instant examination of the blood and the tissue while they are absolutely fresh, are conducive to the attainment of exact and trustworthy results, and to the discovery of the more important and recondite physical relations that most need to be understood in the history of infectious and epidemic diseases. Epizoötics, thoroughly investigated in the light of modern science, can become, and indeed are becoming, the most trustworthy aids to the correct interpretation of the conditions and principles connected with the propagation and the pathological history of pestilential epidemics. The time has come when the medical profession is demanding that the value of this kind of investigation into the pestilential maladies of the domesticated lower animals shall be more intelligently appreciated by all educated physicians who have opportunity for observing

epizoötics. And we deem it due to the medical profession to state, in this place, that not a few of its most learned and practical members are at present pursuing this kind of study in various parts of Europe." Ibid., 1867.

After this digression we will at once proceed to demonstrate these

POINTS OF RESEMBLANCE.

FIRST—THE GEOGRAPHICAL DISTRIBUTION OF THE SOUTHERN CATTLE PLAGUE AND YELLOW FEVER.

As has been mentioned in considering this subject with reference to the southern cattle plague, so far as our present knowledge goes it is not only peculiarly an American disease, but in its primary origin decidedly limited to certain portions of our southern states. We do not know that the disease primarily exists outside of these localities. The same territory is essentially the home of the yellow fever, though it also finds its primary generation in certain parts of the west coast of Africa, South America, and has occasionally occurred in Spain. Wherever these diseases occur outside of their natural climatic localities they are introduced by means of diseased individuals, or by materials polluted by such, but never develop as primary or original diseases in such localities.

Whether or not the yellow fever has ever been spread in the North by apparently healthy individuals from a permanently infected district in the South, as has invariably been the case with regard to the bovine pestilence, is a question I will not take upon myself to decide at present, but there seems to have been occurrences which warrant such a conclusion, as may be seen from the following quotation:

"Experience seems also to have induced general concurrence in the opinion that the disease can be imported by a healthy as also by a sick person from an infected place." Annual Report U. S. Board of Health, 1887, p. 127.

TO SO-CALLED "ACCLIMATIZATION" IMMUNITY.

The idea that acclimatization alone, that is, the mere fact that an individual lives in a certain place a long time, can produce immunity against any disease, is a supreme humbug; the immunity thus acquired is due to constant exposure to the inficiens of a given disease prevailing more or less frequently in such localities, whereby a cer-

tain constitutionalization of the infecting disease producing principle gradually occurs. Both of these diseases are alike in this point. We have seen that many cattle growers living in localities native to the southern cattle plague, the cattle from which are the means of transporting the inficiens from those localities to points where the transporting the inficiens from these localities to points where the disease never occurs as a natural enzoötic, deny its existence among their cattle, but sufficient evidence has been given to show that such cattle do indeed go through a mild form of the disease, and that they continue immune to any serious infection so long as they remain upon such permanently infected lands. However, if cattle from other districts, where the disease does not prevail as a local enzoötic, are introduced into such permanently infected localities, they at once succumb to the disease, and either die, or, if recovering, undergo then what the local cattle men call the "acclimatization fever," and thus acquire the same resistance to further infection that the cattle native to such permanently infected localities have.

If, however, the cattle native to such permanently infected districts are removed from them to those where the disease does not exist, and remain there for a certain period, and are then returned to the original localities, they succumb to the infectious influences with the same certainty that any cattle from non-infected districts do when introduced upon such permanently infected lands.

Now, in these two points, the southern cattle plague and yellow fever deport themselves exactly alike.

On this subject Hirsch says—Geographischen Pathology, p. 241:

"One of the most interesting points in the history of yellow fever is the influence against infection which is demonstrated by the race, nationality, and acclimatization—conditions of the people. At those points in the yellow fever zone where the disease has the character of a more or less constant affliction, whether permanently there or constantly imported matters not, those people who are fresh arrivals, or not acclimatized, are much more liable to infection than those native to such places or that have lived there for a long time, who acquire a greater or less degree of immunity to infection."

It is also true of the yellow fever, as of the southern cattle plague, that if human beings born in, or acclimatized in, such permanently infected localities leave the same and take up their residence in districts where the disease never occurs, under natural conditions, and

after a time return to the first named localities, that they are as liable to infection as if they had never resided in such permanently infected regions.

Hirsch further says on these points:

"With regard to it there prevails the greatest unanimity of opinion among observers, in all places and at all times, so that this peculiar deportment of the people within the yellow fever zone to that disease cannot be better expressed than in the words of La Roche: 'Within the tropics the population consists of two classes; the first, composed of the natives and acclimatized, who, so far as relates to the yellow fever question, live with immunity amid the sick and the dying; the second, of strangers, who are almost inevitably attacked by the reigning disease and perish in a large proportion.'" Ibid.

The above statement from La Roche is altogether too positive; it lays too much stress upon the word "acclimatization" and the ideas connected therewith. A more correct view of the real condition of things is given in the following quotations, which show that something more than mere climatic influence is necessary to produce immunity from the yellow fever or southern cattle plague:

"In the archives of the Havana Acadamy of Sciences a great mass of data is collected to confirm the facts that both white and colored do fall victims to the yellow fever, and are not naturalized, or acclimated, notwithstanding that they have been born in Cuba and have been constantly subjected to its climatic influences." U. S. Board of Health Report, 1880, p. 144.

"In the epidemics at Baracoa, 1876–78, an exceptional feature was that the yellow fever attacked native Cubans with especial vigor and fatality, and not only natives of the towns but also of the adjacent country, and that, as is well known, native residents of the adjacent elevated country are as liable as Europeans to the disease. All yellow fever places illustrate the general rule, that the longer the disease is about the greater is the number of natives attacked." Ibid.

"Cubans born in and residents of the interior, especially of the cool and mountainous parts, are liable to the yellow fever." Ibid.

"Native-born Cubans coming from a healthy district to one where yellow fever prevails are as liable to this epidemic as are unacclimated foreigners, while those that are born and remain a certain number of years in the infected region are exempt from it." Ibid., p. 145.

Every place where the yellow fever habitually prevails proves that the inhabitants gain immunity from the disease." Annual Report U. S. Board of Health, 1880, p. 9.

NATIONALITY OR BREED, IN RELATION TO PREDISPOSITION TO YELLOW FEVER AND THE SOUTHERN CATTLE PLAGUE.

On this point, with regard to human beings, Hirsch says:

"The extent of this predisposition of foreigners is, to a certain degree, dependent upon their nationality—or more correctly expressed, the temperature of their native country. This predisposition is the greater the higher the latitude from whence they come into infected districts; these conditions bear relation not only to the number of cases of disease but to the degree of mortality among the same."

Townsend says:

"The mortality of the vomito to the new-comer from cooler latitudes may be said to be in exact ratio to the distance from the equator of his place of nativity and residence."

Then follow some statistics, giving the proportion of fatality in this respect, which may certainly be pardoned an introduction here:

Accordingly to Barton, in the epidemic at New Orleans in 1853 there were diseased to the thousand people:

Creoles	3.58	per cent.
Foreigners from the West Indies, Mexico, and South America	6.14	"
Foreigners from the southern states of the Union	13.22	"
" " Spain and Italy	22.06	"
" " the middle United States	30.69	"
" " New York and New England	32.83	"
" " the western states of the Union	44.23	"
" " France	48.13	"
" " British-American Provinces	50.24	"
" " Great Britain	52.19	"
" " Germany	132.01	"
" " Scandinavia	163.26	"
" " Austria and Switzerland	220.08	"
" " Holland and the low countries	328.94	"

Ibid, p. 242.

It is to be regretted that no such reliable information is given with regard to the susceptibility of cattle from different latitudes and countries to the southern cattle plague when introduced into infected localities where the disease has its permanent home, but enough is known, as has been previously stated, to show that the general results have been the same in the bovine as the human pestilence.

This is surely a point to which the governments of such states, especially that of Texas, could well give more exact attention in future years.

ACQUIRED IMMUNITY.

Here, again, we find the same conditions existing, with regard to the cattle disease and yellow fever.

Hirsch says on this point:

"The immunity against yellow fever acquired through thorough acclimatization,(?) which, in reality, is by no means absolute, is only arrived at by years of residence in a permanently infected locality, but is most surely acquired by the individual having successfully passed through an attack of the disease. Temporary residence in such a locality is of no value in lessening the liability to infection."

Dutraulau says: "The chances of immunity appear to bear direct relation to the time an individual sojourns in a yellow fever focus; but acclimatization immunity is only acquired by those who have passed through an epidemic period without leaving the country, or such as have completed an attack of the disease."

Dowler says: "It is the resident city Creole, not the country Creole, not the Creole who migrates every summer to New York, London, or Paris, that may hope for as good health as is possible to humanity, while two or three hundred others daily fall victims around him."

Cormillac testifies: "Acclimatization is secured only by residence in infected places during epidemics. Only those Indians and Creoles enjoy immunity who live where yellow fever generally prevails; there is no acclimatization against yellow fever." Ibid., p. 150.

Simons observed the disease in Charleston, S. C., and says: "All persons who have not spent a yellow fever year there are liable to the disease, and it is questionable if they are wholly exempt until they have had the disease."

Rufz communicates the remarkable fact that, "upon the Island of Martinique, which had not been visited by the yellow fever from 1826 to 1838, in the epidemic of the latter year, while many persons became sick, still those who had lived constantly upon the island for a period of from 6 to 10 years generally had the disease in a mild form." Ibid., pp. 242–3.

"With regard to a second attack of the disease in one and the same individual, it is a fact that such is a seldom occurrence, and when it does occur the first attack has generally been a very mild one, or if severe that the individual has been a resident of a northern locality for a considerable length of time."

"The only known mode of acquiring immunity from every other non-recurring disease is to have one attack, and so far as the yellow fever is concerned, while various modes are claimed, this remains the only one so certain that no one whatever disputes it." U. S. Board of Health Report, 1880, p. 153.

With regard to our southern cattle, we know that the same is the case as in the yellow fever, that is, that cattle which have lived in the cattle plague zone acquire an immunity, by having been constantly exposed to infection, because such cattle when imported North, remain perfectly well, though they infect our northern pastures with the pestilence, and thus have been the cause of the loss of millions of dollars' worth of cattle to northern feeders and breeders. This fact is as well established as any in connection with the yellow fever.

The following quotations may be taken as illustrating the universal experiences of stockmen:

"1. That cattle from a permanently infected district which are taken beyond this district to places where the infection does not exist, contaminate pastures, and in that way disseminate the disease among the native cattle in the non-infected district.

"2. That cattle from non-infected districts which are taken into the affected district contract the disease and suffer with the same symptoms as those which contract it in the non-infected district from exposure to the infection of southern cattle.

"3. That the native cattle of the infected districts enjoy an immunity from the disease, and, as a rule, do not suffer from it, either on their native pasture, or when they have been driven into the non-infected section." U. S. Agricultural Report, 1885, p. 250.

We also know, as mentioned previously, that if southern cattle, which the owners say do not have the southern cattle plague at all, are taken North for a sufficiently long period, that they are fully as liable to die or become ill as northern cattle that have never been South are, if taken to such infected southern districts.

RACE IMMUNITY.

The claim, frequently made, that negroes and Creoles, or whites born in constantly infected localities, have a race immunity, seems to be an *a priori* conclusion without any satisfactory positive evidence behind it. Such immunity should rather be attributed to the influences of prolonged and mild exposure to the inficiens rather than to any racial idiosyncrasies.

On this point we have very confirmatory evidence from trustworthy observers.

Shecut says: "Those native children that arrive at the age of nine years, are then considered as naturalized to the climate; but

until this, they stand equally exposed to the disease with strangers or foreigners." Hirsch, p. 243.

"Dr. Mazarrede, who was familiar with and inclined to concur in the view of the immunity of children entertained by numerous distinguished physicians, writes: 'Notwithstanding this, the result of twenty years' experience has shown me to the contrary, and in my own practice I have seen cases of yellow fever in children from one to five years old, and even not over a year old, in whom it has been fatal, and I am now well convinced that children born in Cienfuegos are exactly in the same condition the first years of their lives as are other new-comers, and are just as liable to its attacks. Nevertheless I consider that children are generally less prone to suffer severely, owing to their different conditions of living, and enjoy in this respect the same privileges as the better class of foreigners who suffer little.' Numerous reports from other physicians of exactly the same tenor can be seen in the original." U. S. Board of Health Report, 1880, p. 145.

"Statistical records and general experience unite in proving that sucklings at least suffer comparatively little from the yellow fever." Ibid., p. 152.

"The vast majority of Creole children do undergo attacks of yellow fever; attacks which, however mild, suffice to protect fully as frequently as vaccination limited to a single period of infancy protects from small-pox." Ibid.

With regard to calves from cows raised in the districts permanently infected by the southern cattle plague, we have no such exact evidence, though immunity exists. Experiences in the North, on the other hand, give ample evidence that, so long as the calf is a carnivora, that is, sucks and lives upon milk exclusively, it is not susceptible to infection, though it may take the milk from several cows in succession that have been ill and finally succumbed to the disease.

The following are testimonials of practical cattle men on this point:

"Not in a single instance have I known of a calf dying of the disease."

"Stock of a year old are not exempt from its ravages." N. Y. Transactions, p. 1065.

"I have seen a calf, which is now living and in good health, that was suckled in succession by three different cows which died of this disease in its most aggravated form; the little animal drew its food from them while they were sick, and when the first died it was given to another, and so on. It had never been exposed to infection from Texas cattle. Ibid., p. 1066.

"The only kind of cattle that could be imported into the infected districts with any safety was young calves." U. S. Agricultural Report, 1883, p. 29.

"In an outbreak at Wallace's Switch, Va., 1878, it was noticed that the calves never showed the least evidence of the disease, though suckled by their dams up to the time of their death. Ibid., 1887, p. 41.

This being the case, and as such calves do graze somewhat, and more and more as they age, during the nursing period, it is reasonable to assume that in such permanently infected districts they find occasion to go through a mild form of inoculation before they become absolute herbivora, and their constitutions have become so changed thereby as to render them insusceptible to the inficiens of the southern cattle plague. Furthermore, as there is ample evidence that their parents do go through a mild form of the disease, and as the germs of this disease are morphologically of the same size as those of swine plague, and as I have repeatedly found the germs of the latter disease in the fœtus removed from the uterus of dead sows, is it unreasonable to suppose that intra-uterine inoculation of the calves may occur in a disease where the mothers are constantly exposed to and consuming its inficiens in or with their food and water?

In regard to the degree of exposure of cattle to the inficiens of this southern pest, it seems that the amount of constant exposure should, if we may judge from their manner of feeding and drinking, be much greater in permanently infected districts than that of human beings in similar districts to the yellow fever, if we except the very lowest and filthiest classes of the population. The above remarks would seem to apply especially to the white and mixed races, living in places permanently infested by the yellow fever, for the negro race, on the contrary, appears, according to the testimony, to possess a certain degree of congenital (?—B.) immunity towards the yellow fever, and according to some authorities, the same is true of the Mongolian.

Fenner says: "It is a well-established fact that there is something in the negro constitution which affords him protection against the worst effects of yellow fever."

Doughty says: "In the natives of Africa the constitution appeared to me as secure against yellow fever as a person who has had the small-pox is against its recurrence."

Daniell remarks: "That in an epidemic of yellow fever at Savannah, Ga., not one of three hundred freshly imported negroes acquired

the disease." And Blair says, of a similar outbreak at Guayana—1852–53—"That of 7,890 negro immigrants none contracted yellow fever." Ibid., p. 244.

Of mixed races, however, the case is different.

Fenner says: "The least mixture of the white race with the black seems to increase the liability of the latter to the dangers of yellow fever, and the danger is in proportion to the amount of white blood in the mixture." Hirsch, p. 245.

This congenital immunity of the Negro race seems to me to be a very questionable matter, and entirely wanting in exact evidence, and should rather be looked upon as acquired immunity of constant exposure or recovery observed in the white or mixed races, and by no means a constant racial characteristic. If such negroes live for a long time in a northern and yellow-fever-free country, they are no more immune from attack on return to a permanently infected district than whites from a similar free locality.

Hirsch says: "Africans that have traveled in Europe or in the higher latitudes of America are in no way free from the dangers of infection by yellow fever if they return to permanently infected districts."

Lemprière says: "The above remark has been fully confirmed by my own experiences. In this regard it is worthy of remark that negroes, in Senegambia, Bovista, the coast of Benin and Biafra, at Teneriffe, farther in those parts of North America which are seldom visited by the disease, also in Guayana and Brazil, become more frequently and severely attacked than those living on the Sierra Leone coast, the Antilles and the Gulf coast; that is, in all points where the yellow fever has its permanent home." Ibid., p. 246.

It will thus be seen that while the racial predisposition of negroes living in permanently infected districts is perhaps less than individuals of white or mixed constitutions in the same localities, still that this so-called "congenital" immunity is of a somewhat questionable character, and that even negroes are no better off than other people if they leave such districts and take up their abode in northern localities for any extended period.

There are not any breeds of cattle that have any such racial indolence to infection from the southern cattle plague so far as we know, but we have no scientific data that will allow the expression of a positive opinion upon this question.

CLIMATIC PECULIARITIES OF THE YELLOW FEVER AND SOUTHERN CATTLE PLAGUE.

It has been previously mentioned that these two diseases primarily develop under the same climatic, and hence telluric, conditions, but to make their resemblance in this regard more striking it is necessary to consider the subject with greater regard to details.

Etiologically speaking, in their primary origin, they are more or less strictly limited to the tropical zone.

The rainy period and the consequent moisture seem to present favorable conditions to the eruption of yellow fever, but what part these factors play in the generation of the southern cattle plague it is impossible to determine from the data at command.

According to the records of epidemics of yellow fever at New Orleans, Texas, Louisiana, Mississippi, Alabama, Georgia, Florida, Tennessee, Charleston, S. C., North Carolina, Virginia, Maryland, Delaware, Philadelphia, New York, New England, New Jersey, and Bermuda, collected by Hirsch, the following observations were made:

"During the first four months of the year, January, February, March, and April, the yellow fever has never appeared as an epidemic in either of the above named localities. Only three epidemics are recorded in the early part of May, even in those places, New Orleans and Charleston, more especially marked by frequent eruption of the disease. Even in June the number of epidemic outbreaks is proportionately small. In July and August the outbreaks have been the most frequent and severe. Next comes September with 31 outbreaks, while in October there are only four epidemics recorded and these occurred in the most southern localities bordering on the gulf coast. In November and December there is no record of any fresh outbreaks. The termination of the annual eruptions generally occurs in October and November, and only eleven epidemics have continued into December. The real yellow fever season of the regions named is the summer and early fall months, and the epidemics before June and after September occur generally, if not exclusively, in the most southerly situated states, which shows that the progressive extension of yellow fever from the tropics towards higher latitudes corresponds directly with the augmentation of the temperature in the same." Ibid., p. 248.

During the present year, 1887, we have had a mild epidemic at Tampa Bay, Florida, in the months of October and November, which is reported at end as these lines are written—Dec. 1st.

While we are without data of any amount as to the outbreaks of the southern cattle plague in native cattle in the permanently infected districts of our southern states, still we know that the season for importing northern stock into those districts is the same, as has been shown above, that the yellow fever does not prevail in similar localities, that is, the cold or coolest season of the year. There are localities where it is scarcely safe to import such stock at any time of the year. With regard to the eruptions of the southern cattle plague in our northern states, which, like the yellow fever, is always due to the presence of individuals imported from the southern or permanently infected districts of the country, or to materials polluted by them or from them, the above given data correspond exactly with the experiences of northern cattle men. The disease here occurs most frequently in July, August, and September, and ceases with the appearance of cold weather.

THE INFLUENCE OF FROSTS ON BOTH DISEASES WHERE OUTBREAKS OCCUR IN NORTHERN LATITUDES.

In this regard both of these diseases deport themselves exactly alike.

One peculiarity is common to each disease: an already developed outbreak can and does continue until the mercury descends to the freezing point and keeps there for some little time.

Fearn formulates his observational experiences with regard to this point in yellow fever as follows:

"The cold which merely produces white frosts will not finally check the disease; the temperature of the ground need not fall below 40° F. for this effect to be produced: but to terminate an epidemic ice must form on the surface of the ground." Hirsch, p. 250.

The same is true of the southern cattle plague, and even more cold than above demanded must occur, as shown by the outbreaks at Tekamah, Neb., in the summer of 1887, for there, as well as in Illinois, we had almost a continual formation of ice for the first two weeks subsequent to the arrival of the Texas cattle in the North, and yet very severe outbreaks followed in northern cattle.

These experiences completely contradict the generally received opinion, "that frosts (alone) kill out the disease," for they demon-

strated that even repeated freezing would not do it so long as the germs were protected in the undisturbed pad of manure dropped by the Texas cattle.

Continued and solid freezing for quite a length of time, sufficient to penetrate the whole mass of manure, is necessary to render it non-infectious.

In this regard the circumstances may be more favorable for a northern public with reference to the yellow fever than for northern cattle with regard to the southern cattle plague.

One thing is absolutely necessary in order that freezing may render the germs inocuous in either case. That is, the germs themselves must be destroyed by the cold; the same is true as to trustworthy disinfection; the disinficiens must come in actual contact with the germs to such a degree as to absolutely kill them in order to be effective.

This fact is not by any means sufficiently appreciated by public hygienic officials, and especially by the people.

Too much value is placed upon the deodorizing disinfectants, by which we frequently succeed in but replacing one odor by another, and yet it is assumed that a trustworthy disinfection has been achieved, which is generally followed by disappointment in the result attained.

LOCATION AS AN INFLUENCE IN THE GENERATION OF THESE DISEASES.

The fact that both of these diseases are peculiarly of a local character in their primary origin, as well as secondary eruption (when imported into northern latitudes), has already been noticed. Their etiologically local character is, however, very distinctly marked in the regions where they primarily develop. They are essentially diseases of the lowlands bordering on the sea coast of tropical regions, and in level countries where the drainage is poor and the land not only hot but quite moist. The influence of rapidly growing and decaying vegetation plays a not unessential role in supplying favorable conditions to the nutrition and development of the micro-etiological organisms of both of these southern pestilences. On the other hand, important elevations of the country, with good drainage, a somewhat higher temperature, more exposure to the air, and less luxuriant vegetation, offer conditions unfavorable to the life and development of these germs even in latitudes where the lowlands are permanently infected.

A POINT IN WHICH THESE DISEASES APPARENTLY DIFFER.

The yellow fever of man is essentially a disease of cities and densely populated districts, and beyond that of those parts of such cities where the poorer and most filthy classes live.

The southern cattle plague, on the contrary, is a disease of the plains and open country.

This difference is not, however, as great as it seems.

In such localities the population take little or no care of their refuse material and excretions, and hence are continually supplying the means themselves for the continued pestilential infection of their surroundings.

These investigations have conclusively shown that the fæces of southern cattle are the chief means by which they infect our northern pastures, although the urine undoubtedly plays a part in the role, and as they are held in immense droves on their native plains it is safe to assert that the same materials are the means by which the continued infection of the land and extension and support of the disease is supported in the South, which in reality places these infected and pestiferous places in approximately the same condition as the densely populated and filthy portions of the cities where the yellow fever finds its permanent home in the cities of the South.

It does not seem to me egotistical to claim that the southern cattle plague is now a much more completely and satisfactorily investigated disease than the yellow fever of man, and it is to be hoped that the results of these investigations will stimulate work upon the latter disease.

A very interesting point of comparison to be ascertained is, do the fæces and urine of human beings play the same role in the extension and support of the human as they do of the animal pest? I think there is no doubt about it.

One fact is certain in connection with both of these southern pests:

THEY ARE ABSOLUTELY EARTH DISEASES IN THEIR PRIMARY ORIGIN.

That is, their primary and constant development is dependent upon certain conditions of the earth, which are again dependent upon constant climatic conditions.

The truth of this assertion is made most manifest in my experiments with regard to the manure from diseased cattle.

Not in the manure itself is the danger to be sought, save as it is a vehicle of transportation and infection of the soil. The soil only under suitable climatic conditions offers the medium for the proliferation and biological support of these germs in their full virulence.

I believe the same to be true of the yellow fever. They are not malarial-exogenous, but earthy or fixed exogenous diseases.

The air, as a means of transportation or infection, plays so unessential a role in their eruption or extension as not to be worthy of attention. Southern cattle that have infected our northern pastures and caused severe losses in northern cattle placed upon the same pastures, have been surrounded by northern cattle in adjoining pastures, and have been seen to smell and lick them; the wind "bloweth as it listeth," but in no case have northern cattle in pastures adjoining those where death-dispersing southern cattle have been become infected thereby. In the language of cattlemen: "A fence will keep it off." Hundreds of practical observations have shown this to be a fact. The same is true of yellow fever.

Drake says: "The yellow fever is essentially a disease of the larger and smaller cities; people living in the country, even at a distance of a few miles, have nothing to fear."

LaRoche says: "In the country the disease never occurs, however constant and intimate the intercourse with the infected place may be. None are there affected but those who have taken the disease in the latter, and neither they nor such patients as are brought there from the city communicate the infection to any one around them. On this point the testimony of the profession is almost unanimous." Hirsch, p. 254.

It may be axiomatically asserted that both the yellow fever and southern cattle plague become extended to localities where the disease never occurs under natural conditions either through the agency of diseased individuals or materials from the same; or, with regard to yellow fever perhaps, materials from the infected districts directly polluted with the earth and water from the same.

The manure from cattle cars *en route*, in which southern animals have been transported, has frequently been known to cause the infection of our northern land, and hence our cattle, when there were no southern cattle about, and those that passed the manure had been forwarded hundreds of miles further on.

The previous remarks must certainly serve to demonstrate the true nature of the southern cattle plague as well as the yellow fever as strictly infectious diseases of a local character.

We come now to the determination of the question of pathology of the southern cattle plague and yellow fever, judged by the result of the disease in the infected organism.

From this point of view the

SOUTHERN CATTLE PLAGUE AND YELLOW FEVER ARE EXTRA-ORGANISMAL-INFECTIOUS-SEPTICÆMIÆ.

By this we mean that they are septicæmiæ produced by germs that find their primary origin entirely outside of the animal organism, and that it is not necessary that an organic lesion shall be present beforehand in order that the germs may act, as in all forms of wound septicæmia, such as infectious metritis and other surgical forms of septicæmia. Although not exactly logical, I know, still to distinguish this class of septicæmiæ from the former, I think we may be allowed the term *intra-organismal-infectious-septicœmiœ* for them; although, even in them, the exciting cause, germ, must also enter the infected organism in some way from outside; still the primary cause lies in the wounded surface in the tissues of the diseased organism.

It is not necessary to discuss this subject in all its details here, as the support of this has already found its proper place in the discussion of the specific germ of these diseases.*

While considering this part of our subject, we have called attention to the fact that it seems probable that all those diseases which are caused, or may in the future be found to be caused, by this class of "belted ovoid germs," will be found to belong to this group of *extra*-organismal-infectious-septicæmiæ. It was also mentioned that the swine plague, rabbit septicæmia, the German "Wild seuche," and the hen cholera, also belong to the same class In the case of the southern cattle plague and yellow fever, it is much easier to determine this point than with regard to the swine plague, because secondary lesions are rare in the former, and the lesions are those of a straight septicæmia, while in the latter the secondary lesions are quite extensive, and require earnest consideration to differentiate them from the primary.

*See my report on Swine Plague for a very exact discussion of these points.

That the southern cattle plague is an extra-organismal-infectious-septicæmia, or a blood poison due to a specific germ, is not an original or new idea, though it finds its conclusive evidence through our investigation, is to be seen by reference to the report of the very able physicians already alluded to, who composed the metropolitan board of health of New York city, who also came to the same conclusion.

They say: "Without entering into a description of the changes found in the organs, I would conclude by stating that the disease seemed to be one of blood poisoning, the result of a special germ imparted in some way by Texas cattle." N. Y. Trans., p. 107.

"Evidences of *toxæmia* were early observed to be among the very first symptoms of the obvious stage of the disease." Ibid., p. 1099.

"The symptoms connected with the appearance, posture, respiration, pulse, successive changes in the progress of the fatal or obvious stage of this disease, as all that is indicated by the temperature changes and by the phenomena attending the death of the infected cattle, completely harmonize in the conclusion that all these symptomatic conditions indicate the rapid and fatal operation of a morbid poison or combination of poisonous elements." Ibid., p. 1104.

"As regards the pathological conditions which have have found so constantly as to be justly regarded as attributes of the disease, there were two elements which may not have depended directly upon any primary morbid alterations in the blood; yet it was plain that all these alterations be considered as associated results of some primary morbid poison, that, so far as we could judge by the evidence, operated chiefly upon the blood and the liver in the earliest period of its fatal work." Ibid., p. 1109.

PATHOLOGICAL ANATOMY.

We closed our remarks upon the nature of the southern cattle plague and yellow fever with the assertion that they are both blood-poison diseases due to the action of specific germs bearing as close resemblance to each other as the diseases produced do in their local origin and other phenomena. We have now to give evidence that the diseases are

EXTRA-ORGANISMAL-INFECTIOUS-SEPTICÆMIÆ.

and will begin with the southern cattle plague, and notes from some of our own autopsies:

No. 1.—Red and white steer, two years old, very good condition. Temperature 42° C. Respirations rapid and distressed. Eyes fixed and becoming glassy. Pulse very rapid and weak.

Cut its throat!

Blood of a peculiar reddish color, having more the character and appearance of some coloring fluid than normal blood. It coagulated but slowly on exposure to the atmosphere, and did not become scarlet-red as quickly as most blood does under the same circumstances.

On cutting through the skin of the abdomen the subcutaneous fatty tissue was found to be well preserved. The superficial inguinal glands were intensely swollen and of a diffuse reddish-grey color, the cut surface was moist and glistening, and a red watery fluid exuded from it upon pressure. It may be here remarked that all the other lymph glands of the body were in a similar condition, those of the mesenterium being the most excessively swollen and replete in blood. The abdominal cavity contained about a quart of a yellowish colored fluid. The peritoneum was also somewhat stained with the same color. The outside of the small intestine presented a bright diffuse pink-red color, while that of the large was more greyish.

The spleen was much swollen, and presented a peculiarly variegated appearance, the superficial veins being intensely engorged, and the trabeculæ showing through the capsule. It weighed six pounds. It was twenty-two inches long, eight wide, and three thick in the middle portion, which was more swollen than either end. When cut it was found softened and completely engorged with blood which flowed out of the substance of the organ.

I did not stop to remove the liver, but at once cut out a piece on opening the animal, and wrapped it in three clean napkins that had been in soak in five per cent carbolic acid solution for over a week. I find this the most practical way for taking material home for the inoculation of culture tubes. Any germs that may fall on the outside are destroyed, and the inside will remain perfectly fresh and uncontaminated for several hours.

The whole organ was very much swollen, its edges thick and round, the gall bladder full of a very dark greenish-yellow fluid. On cutting open the liver it was found quite full of blood, the inter-acinous vessels being engorged, the acini were distended and the paranchyma fatty degenerated, which gave to the liver a peculiar greyish-brown-red appearance, which was made still more striking by the markedly injected condition of the gall-capillaries, which could be seen as delicate hair-like lines of a yellow color marking the limits of each acinus.

The kidneys were also intensely swollen, the left one weighing over two pounds. The fatty capsule was found to be more or less marked by numerous hemorrhages and collections of blood. The capsule proper was non-adherent. The outer surface of the organ was of a diffuse, dense blue-black-red (logwood) color; same of cut surface; the medullary substance could not be distinguished from the cortical so far as any difference in color went. The lining membrane of the pelvis was swollen and the seat of numerous hemorrhagic centers; the cavity contained a quantity of coagulated blood which was attached to the mucosa.

The bladder was two-thirds full of a claret-wine-colored fluid; mucosa swollen, vessels engorged, with numerous diffuse and small circumscribed hemorrhagic centers scattered through its substance.

Stomachs: First three comparatively normal. The fourth contained a small quanity of gall-stained, greenish-yellow material. Its mucosa was intensely swollen, of a diffuse livid pink color, interrupted by the engorgement of the larger vessels and numerous hemorrhagic centers of various size and contours; aside from these there were numerous eroded spots with somewhat indurated edges, the base being of a reddish-gray color—not diphtheritic! The whole membrane was covered with a viscid-catarrhal coating, which was stained yellowish towards and in the pylorus.

The mucosa of the entire small intestine was much swollen, and of an intense yellowish-red color, interrupted by numerous darker colored hemorrhagic centers. The solitary follicles and Peyers' plaques were swollen, the latter more than the former. Contents semi-fluid and of a dirty yellow-green color.

The mucosa of the large intestine still more swollen and of a deep red color; the hemorrhages were larger and more frequent, some of them being diffuse and extensive. These conditions increased towards and in the rectum, where the swollen folds or rugæ were very marked; the hemorrhagic condition so increased towards the anus that the entire mucosa was of a dark pink-red color. Contents pultaceous and of a yellowish color, stained with blood on the surface.

The thoracic cavity contained no fluid. Pericardial sack held about two tablespoons of a reddish-colored fluid and covered with petechial spots. Myocardium opaque, anæmic, and friable. Bronchial lymph glands as described for the others. Lungs normal. The mucosa of

the trachea and larger bronchi somewhat swollen and the seat of small petechial hemorrhages. Covering glasses were at once smeared from the fresh blood caught in a sterilized bottle, and also from the liver, and cultures upon agar-agar were also made on return to town. In the blood and also the covering glasses prepared from the liver and blood the germs of the disease were found in great numbers, thus confirming our previous observations. Cultures also developed.

No. 2.—Native steer, two years old. Before killing the animal its temperature was taken and found to be 41° C. It was in fairly good condition; had been ill two days—body covered with ticks—visible mucosa injected and of a yellowish color. Some appetite. Flanks tucked up and fallen in—hair standing; movements weak and uncertain, especially of hind legs. The animal was knocked in the head and throat cut. Blood quite thin, and while it coagulated this process proceeded very slowly—it was a claret-red color and presented the same when running over the fingers—in fact, it seemed more like a red water than blood, not having the usual viscidity of that fluid. On skinning the animal, the subcutaneous adipose tissue was somewhat atrophied and of an abnormal yellow color; the abdominal aponeurosis being also tinged in the same manner. The muscles and flesh of the animal were of a grayish-red color. On opening the abdominal cavity the two first things that struck the eye were the diffuse pinkish redness of the outside covering of the small intestines, and the enlarged and prominent spleen, which was about double its natural size, weighing five pounds. Contents rich in blood, somewhat softened, and of a deep, red-black color. The stomach and intestines were next removed. The first stomach was two-thirds full of grass, with considerable fluid admixed. That of the second stomach was still more fluid, while the third was well distended, but not over hard. The fourth stomach was empty; its mucosa, or lining membrane, was intensely swollen and reddened, with small hemorrhagic centers here and there. This inner surface of the stomach was covered with a viscid mass, which attached intimately to the underlying tissues. The contents of the small intestine was semi-fluid, and not sufficient in amount to fill the tube, that of the large intestine was of a pultaceous character. The mucosa of the entire intestinal tract was swollen and covered with an adhesive viscid coating characteristic of catarrhal conditions. That of the small intestine, especially of the

anterior two-thirds, was of a bright yellow color, being deeply stained with gall. The posterior portion of the small intestine was more red in color, otherwise in the same condition. The glands were much swollen. The liver was swollen, very juicy, of a yellowish, gray-red color. The gall ducts, capillaries, so distended that they could be seen with the naked eye in many places. The acini were enlarged. The kidneys were swollen and of an intense dark, bluish-red color, though the urinary tubes could be seen through it as grayish-white striæ and were much swollen. The dark red condition is due to capillary engorgement. The mucosa and fat of the pelvis of the kidney was of a bright yellow color. The bladder was about half full of a claret-red fluid, which had a specific gravity of 115°. The abdominal lymph glands were much swollen, moist, and in many of them there were hæmorrhagic centers, while others were of a diffuse, dark, bluish-red color. There was a small quantity of effusion in the chest or thoracic cavity. The covering of the lungs and internal surface of the ribs was of a yellowish color and somewhat swollen, while here and there were occasional points of hæmorrhage. The fat around the heart was of the same yellow color. The sack of the heart—pericardium—contained about a tumbler of a yellowish colored fluid. Its muscles were soft, friable, and of a yellowish-gray-red color. The bronchial lymph glands were much swollen and presented similar conditions to those of the abdominal cavity. The anterior, or forward, and posterior terminations of both lungs presented a mottled and a dark red color, and were the seat of numerous centers of fresh lobular pneumonia. On cutting across these points the surface of the cut lung was the same as the outside, but of a glistening appearance, and much water flowed from it on pressure; the vessels between the lobuli were filled with blood, which, in some cases, was coagulated. The above conditions represent those of an accidental complication, and almost entirely correspond to a condition seen in many cases of swine plague. The mucosa of the air passages was somewhat swollen, and tinged yellowish with a few hæmorrhagic centers. The smallest air tubes, especially toward the diseased points of the lungs, contained a watery fluid.

No. 3.—The animal was a native grade steer, two years old. As usual it had withdrawn itself from the herd and stood near a run, alone by itself. On stirring it up, its movements were tottering and very feeble,

unless helped, when it still had strength enough to do some pretty smart running. A close examination of the animal was impossible. It frequently tried to pass manure, but was unsuccessful, except in very small quantities. Somewhat emaciated; hair rough; eyes had an anxious expression. Although evidently a very sick animal the owner did not feel like killing it for our purpose, so that we purchased it for the sum of ten dollars. Shot the animal. Immediately on falling, took its temperature: 42.50 C. Visible mucosa somewhat icteritic, especially prominent in the conjunctivæ of the eye.

Autopsy: No external markings worthy of note. Paniculus adiposus considerably atrophied; blood of the peculiar logwood color and character found in this disease. Coagulated and oxidized very slowly on exposure to air. Peritoneal cavity contained about six quarts of an amber-colored fluid. Costal peritoneum swollen and clouded, and marked by numerous petechial hæmorrhages distributed through it. External surface of the small intestines of a delicate, diffuse, pinkish-red color; some few small hæmorrhagic centers to be seen; serosa of the large intestines clouded, swollen somewhat, grayish-red in color, with numerous petechial spots in its substance.

Spleen exceedingly swollen, edges rounded, trabeculæ visible through the capsule; contents very bloody; weight four pounds.

Liver enlarged, edges rounded, capsule somewhat thickened and covered with vegetation in various places. Gall bladder distended and full of a greenish-yellow fluid. Cut surface of the liver somewhat anæmic. Acini very much swollen, the general color being a yellowish-gray-red shade; opaque, hepatic, intra-acinous veins invisible; gall capillaries presented a beautifully injected condition, and could be seen as the most delicate of yellow hair-like lines taking their course between the acini.

Kidneys swollen; capsule non-adherent; the external and cut surface being a diffuse logwood-red color; the medullary substance was also of a diffuse tint, not so intense a red color. Mucosa of pelvis swollen and replete in hæmorrhagic centers of various dimensions. The urinary bladder contained about a quart of claret-wine-colored fluid; its mucosa was much swollen, the vessels ingested, and many small hæmorrhagic centers were also to be seen.

Nothing need be said about the first three stomachs, but the fourth offered a fine specimen of the conditions common to this disease. It

was empty. The mucosa was very much swollen, and of a very intense deep, but not dark, diffuse red color, which was interrupted in many places by hæmorrhagic centers, and at others there were spots covered with a grayish-yellow material somewhat dry, which on being removed, revealed an ulcerated surface underneath, which was below the surrounding mucosa; the edges of these ulcerations were irregular and swollen. The balance of the mucosa was covered with a viscid adherent coating, the vessels of the sub-mucosa were deeply engorged. and numerous hæmorrhages were to be seen by the careful examination of this portion of the gastric wall; they were generally of a diffuse character; toward the pylorus the mucosa became of a deep yellowish tinge, which extended through the duodenum and into the jejunum. The contents of the latter were semi-fluid in character and of a dirty yellow color; mucosa swollen; numerous small circumscribed hæmorrhagic centers present.

Large intestines: Contents semi-fluid and of a dirty yellow-green color. Mucosa swollen and of a light pink color. Numerous hæmorrhages scattered through it, which became more profuse and diffuse as well as extensive as one approached the rectum; toward the anus the entire mucosa was of a dense dark-red color, and very much swollen; the crests of the rugæ were marked by many small centers covered with a dry yellowish-gray material, and of the same character as those seen in the stomach.

No effusion in thoracic cavity. Lungs comparatively normal. The myocardium was opaque, anæmic, yellowish-gray-red in color, and friable. Lymph glands of entire body intensely swollen, œdematous, and a diffuse pink-red color.

From these autopsies sufficient evidence can be easily drawn to corroborate the assertion that the southern cattle plague is an infectious septicæmia. The disorganized condition of the blood, the numerous hæmorrhages, the engorgement of the spleen and lymph glands, are all specific to such a complication, while the parenchymatous changes of the liver, kidneys, and heart, as well as the muscles, are all secondary lesions, finding their cause in the fever produced by the action of the specific poison upon the caloric centers. Hueppe has come to the same conclusion in regard to the German swine plague, rabbit septicæmia, hen cholera, and the "Wild seuche," all of which are diseases caused by a

specific, but not actually identical, bacterium of the ovoid belted group, but has used the term "*Septicæmia Hæmorrhagica*" to express his meaning. Objections can be raised against this nomenclature, because all forms of septicæmiæ are more or less hæmorrhagic in character, and hence I think the term selected by me, "*extra-organismal-septicæmiæ,*" more clearly expresses the true nature of this class of diseases.

The primary and secondary lesions above referred to, occurring in our southern cattle plague, would answer almost, if not equally well for the yellow fever in man, as may be seen from the following:

NECROSCOPICAL LESIONS IN YELLOW FEVER.

"The heart appears soft and pale; the myocardium is very friable and in a state of fatty degeneration; sometimes it may appear normal. The pericardial sack often contains a considerable quantity of a serous fluid, which is of a yellow, or yellowish-red color. The blood in the ventricles is sometimes fluid, and at others coagulated and varies much in color and reaction. The coagulum, which frequently extends into the larger vessels, is stained yellow, as well as the endocardium and intima of those vessels. The organs of respiration show no characteristic changes, but hæmorrhagic infarctions are sometimes present in the lungs and ecchymoses in the pleuræ. In some few cases excessive transudates are present in the pleural cavity.

"The most important and characteristic changes, however, are to be found in the organs of the abdominal cavity. The mucosa of the stomach and small intestines, as well as the œsophagus, is invariably in a swollen and catarrhal condition. The individual vessels, especially the veins, are much engorged; hæmorrhagic erosions are frequently present in the stomach; ulcerations seldom. The lymph glands show no constancy in their changes. The liver does not present any very marked changes in volume, but is frequently somewhat enlarged; it varies between a bright yellow, nankin, butter, or straw, to a milk, coffee-brown color. This yellow color is frequently diffuse, but at other times varied. The liver cells are granulated, their nuclei being indistinct and very replete in drops of fat; they show no change in form. The entire parenchyma is in a state of fatty degeneration."

(These fat globules, and even the granular detritus, often become of a bright yellow color, due to gall staining, both in yellow fever and the southern cattle plague. It will be remarked that no mention is made of the engorged or injected condition of the gall capillaries in the liver in yellow fever, which is a constant accompaniment of the cattle plague of our southern states.)

"The spleen is not very much enlarged, but is of a dark color and very full of blood; sometimes the parenchyma is friable.

"The kidneys are swollen, and the parenchyma in a condition of fatty degeneration. (Other authors speak of acute "Brights" as being an almost constant condition, and uræmic phenomena one of the closing scenes of this terrible malady). The mucosa of pelvis is swollen, catarrhal, and the seat of ecchymotic hæmorrhages; mucosa of the bladder is in a similar condition." Haenisch, Ziemssen's Handbuch, p.p. 496-7, vol. 2, 1876.

Without desiring to enter upon any more detailed account of the lesions in these diseases, still I wish to call attention to a passage from Cornil-Babes, a portion of which I do not find in the other authors at my command. They say:

"A few days after the remission (which most authors speak of in the yellow fever), the symptoms of a general parenchymatous degeneration commence to show themselves; at the same time the vessels of the mucosæ and the kidneys become changed; these parenchymatous changes are marked by a severe type of icterus, and by albumen and cylinder casts in the urine, as well as internal hæmorrhages, petechiæ in the skin, by the black vomit, and hæmaturia." Les Bacteries, p. 346.

It is the hæmaturia to which I especially desire to call attention, as it is also a very constant phenomenon in the southern cattle plague, and, therefore, generally looked upon as a more or less characteristic of that disease. These authors are the only ones who call especial attention to this phenomenon in the yellow fever, and hence it is valuable, as it shows another striking resemblance in the two diseases. I would not have it assumed that hæmaturia should be looked upon of itself as being characteristic of the southern cattle plague, as it is sometimes wanting, and also occurs in anthrax, as well as a peculiar complication which occurs in cattle, which is commonly termed "red water," but which has none of the other characteristics of the southern cattle plague, especially as regards origin. The log-wood, red-colored kidney, due to diffuse capillary engorgement, which seems to be, indeed, decidedly peculiar to the southern cattle plague, does not appear to occur in the southern pestilence of man. This seems to be the only pathological lesion in which these diseases decidedly differ. The gastric and intestinal lesions are the same in each.

With regard to the cattle disease, it was once looked upon as anthrax, but aside from the engorged spleen, it has scarcely a pathological lesion of that malady. The blood, instead of being dark and thick like tar, is thin, and, as said, more like a logwood dye than real blood. Again, one misses the œdematous condition known as "gelatinous infiltration," which occurs where large masses of connective tissue exist, and which is very characteristic of anthrax, and above all, the Bacillus anthracis is missing. Anthrax is also a disease of almost any climate, and occurs more frequently in isolated cases, with us, while the southern cattle plague is a foreign disease in our northern states, and when it occurs can always be traced to the influence of southern cattle. These remarks have been appended, because it does not seem necessary to enter upon any discussion of the symptomatology or diagnosis of the southern cattle plague in a paper of this kind. For, given the presence of such cattle within the limits in which the northern states are free from frosts, and an outbreak of a highly fatal disease among cattle that have been exposed to such an influence, characterized by want of appetite, cessation of rumination, constipation, and hæmaturia, and every practical cattle man in the North knows full well that he has to do with Texas, or southern cattle plague.

PROPHYLAXIS.

The prevention of the southern cattle plague naturally divides itself into two distinct heads, viz.:

1. Southern cattle plague in the northern states.
2. Southern cattle plague at home.

Let us consider the first of these questions.

As has been shown, and as is equally well known, every outbreak of the southern cattle plague, in what we may call the northern states, is, or has been, due to the direct importation of southern cattle into the same. It matters not to the question whether an outbreak in the North has been due to the infection of our northern pastures, or roadsides, directly through the presence of southern cattle upon the same, or whether such infection has been due to them indirectly, that is, through their manure, which, as has been shown, has been the cause of several outbreaks of this disease in the North, even when the southern cattle had never, themselves, been on the infected lands, but

through manure that had dropped from or been cleaned out of cars in which such cattle had been transported.

This again gives us two other sides to the question of the southern cattle plague in the North:

1st. The cattle themselves.

2d. Their excreta dropped or cleaned out of the vehicles of common carriers—railroad cars.

As to the first, viz., the cattle themselves:

That these southern cattle have become well graded up is a well-known fact; that they fatten easily, are cheap, and hence profitable articles to the cattle feeder, is equally well known, as is the fact that they themselves seldom if ever die from the effects of the very disease which they introduce, and which is so fatal to northern cattle if exposed to the infectious principle planted by the southerners upon our northern fields.

These cattle being cheap, well formed, and easily fattened, it is a question of national economy, as well as public health, that some means should be found by which they can be handled in the North without the present danger to the cattle native thereto. Any question which bears so closely upon the food supply of the nation cannot and should not be neglected by the representatives of the people, as has been too much the case heretofore.

The danger, however, which the importation of these cattle is fraught with to northern stock has led to all manner of really necessary restrictions upon their importation, which thus keeps away from the consumers a considerable quantity of one of the essentials of life and tends to enhance the value of that which they do have. Hence, some means must be found to overcome this evil.

As the case now stands, these southern cattle are not, or should not be, allowed to be imported into, or transported over, a northern state between the 1st of March and 1st of November of each year, which are the very months in which they can be most easily and profitably fed for the winter market, the very time when the public consumes the most meat, and when it should be as cheap as at any other.

Now, there is but one way out of this difficulty at present, and that is to *quarantine these southern cattle at some given point outside of the most northern limit where the disease exists, for a period, to be determined by experiment, which will insure their having freed themselves from all*

disease-producing elements. My present idea is that thirty days will be found amply sufficient.

Such a quarantine station should be supplied by the National Government, and could equally well serve as a station for experimentation and study. Such a station should be supplied with all the necessary means for cleansing the cars in which such southern cattle had been imported and for their thorough disinfection. Unfortunately they cannot be "boiled"! as a certain astute state veterinarian advises owners to do in order to disinfect their harnesses and robes when they have been in contact with a horse that has been afflicted with glanders, but they can be thoroughly cleansed and disinfected nevertheless.

It is not my purpose at this time to enter upon the discussion of disinfection, as it is proposed to make that the subject of exact research at some future time, but let me here express my determined objection to any faith in the anti-germicide value of sulphur or chlorine smokings. They are absolutely valueless, as well as the majority of those (so-called) disinfectants which replace a bad odor by a worse and more dangerous one. I allude to carbolic acid, which is too expensive to be used in the wholesale manner necessary to guarantee safe results.

A rule of all trustworthy disinfection must be, that the anti-germicide shall be used in such a manner and in such a strength as to come in contact with and actually kill every germ present. For this purpose nothing is so good as freshly prepared whitewash, made from unslacked lime, to which is added one part in five hundred of crude corrosive sublimate. This mixture is not only cheap, but effective!

Any other system of quarantine seems to be impracticable. Such cattle cannot be quarantined by the respective states, within any point on their own territory, simply because, in many instances, they could not be transported to such a state on account of another state, the territory of which they would have to pass over, which procedure is forbidden by law in order to protect the cattle interests of such states.

Hence, a national quarantine, as suggested, is the only feasible way out of this difficulty.

Otherwise all southern cattle must be forbidden importation north of the plague limits during the period previously mentioned.

Now as to the second question:

THE RESPONSIBILITY OF COMMON CARRIERS.

Sufficient evidence has been given to show that the manure and refuse from cars in which southern cattle have been transported has been the cause of outbreaks of this disease in the North, hence some means should be found which would guarantee the proper cleaning and disinfection of such cars.

Here, again, it seems as if it would be impossible to have this securely carried out under state authority, and that a national law should be made requiring that all stock cars should be cleansed and disinfected every time that they have been used to convey live stock, no matter where from, or where to, and only at special localities on the road, and always under the supervision of a government inspector. While causing some inconvenience, still this procedure is not the terrible "mountain" that many would be inclined to make it. Such cars should be at once sealed by the depot master at any small station where live stock may have been unloaded, and conveyed to such a disinfection station, of which there should be several on all the main lines, supplied with a house, a boiler, and the means of forcing hot water, so as to wash the cars, and a place where the offal could collect and be disinfected. This is done very completely in Germany at little inconvenience to the railroads.

The experiences in Illinois and Nebraska during the summer of 1887 have conclusively shown that the former limits of importation, which extended to April 1st, cannot be longer adhered to, as well as that the formerly accepted opinion, that one frost would check all danger to the northern cattle from any influence planted by the southerners on our pastures, was a most serious error, as quite extensive and many successive freezings occurred after the arrival of those southern cattle in the North.

Wherever such southern cattle are imported into the North, it seems as if it would be advisable to make such arrangements as would guarantee their being unloaded direct into the pasture or lot where they are to remain, in every case that such arrangements can be made by the railroads, and that where this cannot be done means should be found to have them driven direct to such places, and as much as possible over such ways as were not trodden or grazed upon by northern cattle.

Again, it seems as if stock cars should be made tight at the bottom, so as to preclude all possibility of manure and refuse dropping from them in the free and easy manner it now does, and that the only place where it can be taken out should be the door. The bottom, side, and end boards could be made movable, however, if suitable means were found to fasten them securely while in transport.

We now come to the question of the

PREVENTION OF THE SOUTHERN CATTLE PLAGUE AT HOME.

Of this we cannot say very much at present. There is no question to my mind that the smaller the grazing grounds become the more they have to be reduced to the so-called tame grases; the more they are cultivated and drained and exposed to the air, the sooner will it be found that these factors will exert some favorable influence towards checking the development of the specific germ of this disease and the pollution of the grasses the cattle feed upon.

Again, there is no question that this disease can be prevented by inoculation. This statement is positively proven by the fact that these southern cattle, though carrying in them and dealing out a deathly principle to our northern stock, never become diseased, even though grazing among the sick and dying. Such a procedure has, however, little or no value to the northern stockman, but may be made available to those of the South in regard to cattle purchased in the North. They could be inoculated before their being taken to the infected districts of the South.

These questions have to be settled, however, in the South, and with the assistance which the National Government has given and is to give the respective states for inaugurating such researches as we have published here, it would seem as if the natural history of the germ of this disease should soon become better known and many practical points of value ascertained.

ARTICLE II.

THE "CORN-STALK" DISEASE IN CATTLE.

ARTICLE II.—*The "Corn-Stalk" Disease in Cattle.*

THE "CORN-STALK" DISEASE IN CATTLE.

The name, *"Corn-Stalk" Disease*, though not correctly expressing all the facts, has been selected for this pathologically new and scientifically unknown disease in cattle because practical experience has shown that its eruption is generally connected with the turning of cattle into stalk fields in the later fall and early winter months, as a sort of lazy man's method of gleaning up the remnants. As will be conclusively shown, and in conformity with the actual facts, it is not the corn stalks, but the leaves and tender top-shoots, which indirectly cause the peculiar disease in cattle, (and other herbivorous animals?) of which we are about to treat. Any one who carefully observes the cattle when turned into such fields will see that they seldom meddle with the hard, dry, bulky stalks, but, as they pass along, that they pull off the leaves and the tender top portions of the stalks. The same complaint has also been attributed to the smut in corn; but a little reflection combined with observation would show that this idea in entirely groundless, as has been demonstrated by experimentation. Another equally absurd idea, to which we shall soon allude in detail, is that lack of water or salt is the cause of this malady. How long this disease has afflicted cattle in the United States it is impossible to determine, nor can we make any estimate whatever as to the amount of loss it annually causes the farmers of the great corn-raising states of the West, though it is by no means limited to them. Still it can be safely said that this malady causes more loss in cattle to the Western farmer than all other causes combined, not excepting abortion.* The more one becomes engaged in the scientific investigation of these questions, the more does he become convinced of the great urgency existing for the collection of exact statistics on the losses in our live stock annually, and disgusted with the farce played in nearly all our states by

* With regard to the prevention of abortion in cows, German investigators have shown that the injection, under the skin, of a two per cent solution of carbolic acid, once every fourteen days during the fifth to the seventh month of pregnancy, will prevent this disaster in cows having acquired that tendency.

the totally incompetent lay cattle or live-stock commissions, and the utter inadequacy of the present systems of education in the veterinary schools of the country to prepare men for these most responsible, and, if adequately filled, highly honorable positions. It should never be forgotten that it is the qualifications of the individual which make a position honorable, and not the position the individual. Persons totally ignorant of the advanced position of modern hygienic medicine, and the methods of exact scientific investigation, must of necessity be utterly incompetent to comprehend the true responsibilities or nature of the work connected with the preservation of our live stock from preventable diseases. As is well illustrated by the disease in question, such persons are totally dependent upon the musty and antiquated ideas of earlier writers for their knowledge, and absorb it unreflectingly as "gospel truth," according to the historical reputation of the author. They have neither the knowledge, ability, or desire to think for themselves. To such empirics, the "has been" must always be true. They never think to weigh it by the living present. Facts presented to their eyes are utterly ignored, while the truth is entirely hidden by a mighty respect for the ignorance of the past, when covered with the glamour, sometimes, perhaps, deserved, of authority.

So much for an introduction!

The first allusion to this disease which I find in the literature at my command is an article by

PROF. JOHN GAMGEE ON THE ILL EFFECTS OF SMUT IN THE FEED OF FARM ANIMALS,*

who says: "The opportunity presented itself last fall for an inquiry as to the manner in which the smut which attacks plants, may affect animals. The latter part of 1868 was, throughout America, very wet. A large amount of corn became smutty — that is to say, was attacked to a very serious extent by *Ustilago maidis*, and reports reached me from the West and South that cattle were dying in large numbers from a mysterious malady, the origin of which was unknown. From Mills county, Iowa, I was informed, late in November, that about the 12th of the month there was a fall of snow six inches deep, and that the cattle, which usually roam at large on the prairies, were taken in by all the better farmers who had their corn gathered, *and turned into*

*Report of the Commissioner of Agriculture on the Diseases of Cattle in the United States. Washington, 1871, p. 73.

the stalk-fields. In about eight days the cattle began to die, all presenting the same symptoms. My informant lost four out of nineteen head, in fourteen days.

"Personal inquiries among gentlemen from different parts of the United States enabled me to trace the malady in Western Virginia, Illinois, and the Carolinas. It is much to be regretted that accurate information as to the extent of the losses and localities affected cannot be secured.

"There are other circumstances under which cattle die from eating corn. The stalks, very late in the season, are apt to become very hard and indigestible; and without a free admixture of grass, which the early frosts kill, they are apt to cause indigestion and death. This is an observation that has often been made in America. The facts published with regard to the prevalence of a malady among cattle in America, caused by eating smutty corn, are very few. If, however, the real cause of so-called dry murrain had been correctly recorded, there would be no difficulty in demonstrating that the condition of the corn fields has had much to do with developing this disorder.

"The Department of Agriculture has received information of the death of cattle from eating smutty corn, in Hampshire county, Massachusetts. Also from Whitley county, Indiana, where seven head of cattle out of fifty died, 'probably from smut in the corn field in which the herd ranged.'

"From Storey county, Iowa, it is reported that 'last November a disease appeared among herds recently turned into corn-stalk fields. The disease is evidently the dry murrain. Post-mortem examination showed the mucous membrane of the stomach to be highly inflamed. It is evident that the disease is generated in the stalk fields, and probable that it is produced by gorging the stomach when first turned into the stalks after being confined on the wild, frost-bitten prairie grass, and lack of sufficient water.' A few cattle died of dry murrain in Audubon county in the same State, 'supposed to be caused by smut in the corn-stalks.' A few head were lost from the same cause in Calhoun county, and many are reported to have died in Marshall county. We are, however, informed from Sac county that many cattle died in December—cause unknown—'some supposed from eating smutty corn, but that has been disproved.' It is to be

regretted that more is not stated with regard to the reasons which led persons to doubt the effect of the smutty corn. Even in New York little credence was given to the action of smutty corn at first; but careful inquiry proved that after all it was the cause of the dry murrain in the fall of 1868. From Dakota county, Nebraska, we learn of dry murrain from the same cause, whereas from Shoshone county it is reported, and no doubt correctly, that the same disease has been noticed in cattle fed on prairie hay cut after frost."

Let us now subject these views of Mr. John Gamgee to critical analysis. It is self evident that the above statements are all of an *a priori*, that is, without proof, character; that Mr. Gamgee had actually neither practical knowledge of, nor experience with, the disease of which he was writing. Yet it seems highly probable that he is the responsible authority for the prevalence of these ideas in the minds of the American veterinarians of to-day, and in our literature. In fact, Mr. Gamgee accepted and but repeated the idea prevailing more or less among cattle men of the time, who, on observing disease sometimes occurring in their cattle after being turned into stalk fields in the fall and winter, and seeing, or perhaps supposing, smutty corn to be present, and nothing else to attribute the trouble to, jumped to the conclusion that the smut must of necessity be the cause. They did not, nor did Mr. Gamgee, stop and think that hundreds of cattle, in adjoining fields, or those near by, were also turned into corn-stalks, and at the same time of the year, and still did not become ill, nor did they, or he, go into those fields and examine for the existence of smut. A little common sense and critical consideration, combined with exact observation, should demonstrate to any practical man that dry corn stalks, of themselves, could never have caused the trouble, nor could the smut. It is a well-known fact that many cattle acquire a decided taste for corn smut, and even seek it out, and no evil results have ever occurred therefrom.

Now a word as to the term "dry murrain."

As has already been noted in the previous communication, it is one of those meaningless terms which has crept into veterinary literature from the days of crude empirical ignorance, and been fostered by men whose education should have taught them better than to use it. The word "murrain" itself is an old English expression used to denote the existence of any malignant disease among a considerable number

of cattle, and was once especially used in connection with the rinderpest and the contagious lung-plague. It has no specific meaning. The word "dry" has been prefixed to it simply to designate the fact that the contents of the third stomach are in a more or less abnormally "dry" condition. We will refer to this question again later on. Let us now turn to Mr. John Gamgee's testimony upon the "smut" question.

SMUT NOT THE CAUSE OF THE DISEASE.

Mr. Gamgee says that he was "Anxious to try some experiments on the action of pure smut on cattle. I employed a negro, in January, 1869, to go into the country and collect for me a large quantity of pure smut.

"It was rather late, and the rain had washed most of it off the still-standing stalks, but I obtained *forty-two pounds of excellent smut, free from adventitious matters.* On the 26th of February I purchased two cows in good health, aged about seven years. One cow was fed thrice daily one and one-half pounds of corn meal and three ounces of smut, with as much cut hay as she would eat. The second had the same allowance, but wet. On the 7th of March the amount of smut given in each feed was increased to six ounces. The cow fed on dry food lost flesh. On the 15th of March the dose of smut was increased to twelve ounces three times a day. The cow on the wet food increased in condition; the other one lost. *In three weeks the two cows consumed the forty-two pounds of smut!!* They had a voracious appetite the whole time, and the only indication of a peculiar diet was a very black color of the excrement."

"*Forty-two pounds of smut consumed by two cows in three weeks,*" *or twenty-one pounds to each cow, with no evil effects whatever!!*

Does any sane person for a moment think that any cow or steer turned into a stalk field would possibly consume twenty-one pounds of smut in three weeks' time? The smut would have to be most fearfully plenty, and the field very large, which would yield twenty-one pounds of smut in the late fall or early winter months, and the cow most excessively busy. In fact, so busy would she have to be collecting that twenty-one pounds of smut in three weeks that she would have no time to collect any other food, and would probably die of starvation before she completed the job. If twenty-one pounds of smut had no ill effects upon either of the two cows when forcibly

fed upon it, we may be very sure that smut never killed a bovine when turned into a stalk field; and hence, Mr. John Gamgee's testimony on this matter is absolutely worthless, from a positive point of view. As to "frost-bitten grasses" having killed cattle, were that a cause, every year more than half the cattle in our Western States would have to die. Such statements, or reasoning, from persons claiming education and practical experience, are absolutely sickening; yet they find credence in the minds of many veterinarians, who, having no powers of observation or reflection, and being absolutely devoid of common sense, repeat these mythical statements in the ears of anxious cattlemen who are obliged to turn to them for advice.

It has been said that these misleading teachings of Mr. Gamgee have made their impression upon veterinarians and veterinary literature. In proof of this assertion, the reader may turn to the "Second Biennial Report of the Board of Live Stock Agents" of Nebraska, p. 23, where he may read:

"CATTLE IN CORN STALKS.

"During the months of October, November and December in each year, many reports are received by telegraph and mail that cattle are dying of some unknown disease. The State Veterinarian and assistants have investigated a great number of these reports, and find that in every case the cause of trouble was the feeding of corn-stalks left standing in the field. More cattle have died of this in this State than of all other causes combined. The disease is so easily prevented by good management and proper feeding that the loss from this cause should be very small.

"Corn-stalk fields are a very dangerous range for cattle, more especially so when the season has been favorable for ripening the stalks completely, and which has changed the starch and nutricious matters of the stem and leaf into an indigestible fibre, wholly valueless as food; and with a herd of cattle turned into such a field from off a dry-grass range in the late autumn, there can be but one result: Overgorging of matter which cannot be digested, impaction, and the loss of many valuable animals.

"The symptoms vary, but in nearly all outbreaks of this trouble some of the following symptoms are noticeable: In some cases the animals are wild, with head erect and eyes protruding, and a disposition to go where they please or to attack anyone who may come in their way. Others stupid, dull, with low-hanging heads, more or less salivation, wabbling of the hind parts, knuckling of the fetlock joints behind, inability to get up when down, stumbling, great nerv-

ousness, twitching of the muscles, loss of sensation, loss of appetite, rapid breathing, quick pulse. Many die. All of these symptoms are not noticeable in the same animal, but more or less of them are. As a preventive, cattle should not be turned into a field of dry stalks until after they have satisfied their hunger, and then only for a short time, particularly if the weather has been dry for some time previous. Allow plenty of salt and an unlimited quantity of water."

The above remarks were really intended to nullify certain conclusions of the writer's, which, unfortunately, were and are based either upon exact experimentation or very close observation. Accompanied by many similar clinical symptoms we have, in Nebraska, anthrax, which may occur at any season of the year; a disease which is generally spoken of as "Hydrophobia in Cattle," and which, according to popular rumor, and often most positive assertions, is always connected with the presence of a "mad-dog." There may be still another cattle disease which apparently occurs in July, August, and September, in which the symptoms are much the same as those presented by animals having the corn-stalk disease, though the pathological lesions are different. This must be still another disease, though it may yet be shown that it is the "Hydrophobic" malady. My having differentiated these diseases from anthrax, not one of which these State Veterinarians could possibly diagnose correctly, caused them to fall back on historical authority to decide the point at issue, and to attribute these diseases to one and the same cause—that is, corn-stalks.

In the month of February, 1889, a very extensive outbreak of the "corn-stalk disease" occurred in the vicinity of Fremont, Nebraska, the following accounts of which appeared in the daily papers:

"FREMONT, NEB., February 7.—John Delaney, a farmer living in Elkhorn township, five or six miles east of Fremont, is suffering the loss of a large number of cattle from his herd by a disease which puzzles the veterinarians. He has lost fifty head to date. Yesterday he determined to investigate the cause of the fatality. He summoned State Veterinary Osborn and Dr. Dulin, who made an investigation. They dissected several head of dead animals and found the symptoms exactly the same in all of them. The third stomach was packed full of dry, hard food, and the surrounding organs and tissues were badly inflamed and feverish. The doctors were unable to exactly diagnose the case, but they gave it as their opinion that it was not a contagious disease. The animals when first affected will bellow in a low, hoarse manner, shaking their heads. Within twenty-four hours after this they invariably die."

The *Tribune* of Fremont, of the 8th inst., however, gives us the results of the investigation of the "Live Stock Commission," as follows:

"Dr. Osborn, State Veterinarian, was seen this morning. Speaking of the fatality among John Delaney's cattle, an account of which was given in the *Tribune*, he said that Mr. Delaney and many of his neighbors felt sure that the disease was a contagious one, but there was no reason or theory for any such supposition.

"'There is no contagious disease known to veterinarians which affects the third stomach of the cow,' said the doctor, 'and that was the seat of the trouble in every case I examined. The third stomach, or manifold, was packed with dry food, which, taken in the fingers, crumbled like flour, and was so light it would blow away like chaff in the wind. The whole cause of the trouble is the condition of the food the cattle eat. The hay and corn-stalks are excessively dry, owing to the very dry fall and winter; and the cattle not having sufficient quantities of salt and water, congests are found. There has been some loss on the Hershey ranch, and a post-mortem examination of the animals disclosed the same conditions. Mr. Hershey had lost sixteen head at the time I was there a few days ago.'"

Mr. Delaney writes me that his actual loss was:

10 steers valued at	$300
12 calves valued at	120
39 cows valued at	974
Total loss, 61 head valued at	$1,394

It will be at once seen that Dr. Osborn followed the authorities in his conclusions; but I will show that there were other lesions present in the cattle than that "the third stomach was the seat of the trouble in every case examined." I will show that this condition of the third stomach was but the result and expression of another and most specific malady; that it was not diseased, but that its contents alone were the language of something else of an entirely different character.

This brings us again to that ridiculous term, "dry murrain." It will be seen by reading Mr. Gamgee's remarks, and those of the State Veterinarian of Nebraska, that neither one considers the "corn-stalks" to be, of themselves, the chief cause of the malady, but that insufficiency of water supply plays a still more essential role in its etiology, to which these later authorities have added want of "salt" also.

More "pure theory," as the people call it, more unfounded *a priori* reasoning or conclusions than the above, could not possibly emanate from the human brain. A moment's reflection, or an acquaintance with the anatomy and physiology of cattle, should teach any one that such a "dry" condition of the third stomach is absolutely impossible in cattle, under the conditions mentioned. Any one should know that the gastric arrangement of bovines is similar to that of the camel, the first stomach being the recepticle for vast amounts of both food and water, thus supplying a surplus of such materials over the almost hourly demands of other animals in comparison, which fact has been taken advantage of by man in countries where extensive desert tracts prevail, barren of either food or water, the camels being able to go several days without either, but more especially water, without any intolerable inconvenience. It has often been recorded by travelers that when the water of the caravan had given out, that a camel had been killed in order to get a supply of that vitally necessary material to the preservation of human life. In this regard camels and cattle are exactly alike, and under such conditions as prevail in our agricultural districts, it is beyond the range of possibility that cattle should go so long without water as to produce any effect whatever upon the dryness of the contents of the third stomach or manifold.

The dryness of the third stomach mentioned by Dr. Osborn was not due to an insufficient supply of water, far less a lack of salt, but to an extremely high and prolonged rise of temperature in the animals, in connection with an acute blood infectious-septicæmic disease, which condition would have been at once revealed by the use of a thermometer upon some of the diseased (living) animals; and which any competent person would have immediately recognized in the character of the necroscopical lesions.

LACK OF WATER OR SALT CANNOT POSSIBLY CAUSE THE CORN-STALK DISEASE.

Having previously shown the impossibility of these two factors playing any role in the causation of this disease from theoretic, anatomical and physiological grounds, I wish now to present practical evidence of the correctness of my assertions from the observations of a farmer, in the form of one of the most remarkably intelligent and observant letters it has ever been my fortune to receive from a live-stock breeder.

Mr. Samuel McKelvie, of Fairfield, Neb., wrote me under date of April 1, 1889:

"During the past fall and winter I have lost nine head of cattle from the stalk-field trouble, whatever it is. I have lost all of these from one field of forty acres, and I have noticed closely, and have thought of different causes and theories; but the last one that died knocked all out of me. I am at a loss to know what the cause is, unless some disease is in the field. I will give you the particulars as near as I can, and if it is not asking too much of you, I should be very glad to hear your ideas about it.

In November last I commenced feeding my cattle (about 40 head) shock corn from this field of forty acres of which I have spoken. They then ran in a pasture of two hundred acres. One day I noticed them gathered in a bunch in the pasture, and on investigating found one of the number dead and swollen tight; it was a calf that had been weaned some three weeks, and in excellent condition. I did not examine it. In about two weeks, or about the last of November, I got the corn all gathered out of said forty acres, of which about three acres had been cut up in shock and the rest left, the corn being shucked off. On Sunday I turned my cattle in about one and a half hours in the forenoon. The balance of the day they were in a lot with straw (oat straw) in a rack, with plenty of water and salt in the yard, so they could get it if they wished. On Monday I turned them back in the forenoon, in said field, about two hours. Tuesday turned them in about two hours in the forenoon and two hours in the afternoon. Wednesday morning I left home early with orders to turn the cattle in the stalks same as on the day before. My little boy went out after breakfast and found two dead and one sick. The hired man proceeded to doctor the sick one and while at work at it noticed another sick. The first one soon died. The second lived until the next day; stood up all the time; must have dropped dead off his feet. We saw him a few minutes before death standing up. They did not bloat until just at the last, and some did not bloat any. This last steer grunted and seemed to be in great misery. Of the four that died, three were yearling steers and one a small two-year-old. Now I thought if there was plenty of water in the two tanks in the yard, that the larger cattle had kept these away until the dry food packed in the stomach and killed them. So I turned those that were left back the next day, two hours in the forenoon, and brought them into the yard and stayed with them until all drank; turned them back in the evening for the same time and saw that all drank when they came out; next day I turned them back, or looked through them before breakfast, and they were all right; sent the boy after breakfast to turn them into the stalk-field. While in at breakfast one of the best two-year-old steers had been taken sick and left the rest that were about

the straw rack, and went below the barn and laid down. In about two hours from the time the boy went to turn them into the field, I went to bring them in and water them, and on going to the barn to get a horse, noticed the one above spoken of lying below the barn almost dead. He would stretch out and tremble and grunt, and could only raise his head off the ground to his side. I sent for the cattle to the field at once, and stayed with the sick steer. When the cattle came in I counted them, and found one short. I sent back to the field, and the report came back that another good two-year-old steer was almost dead in the stalk-field. I went up and found him in about the same condition as the one just described, and they both died in about two hours. When I came back from the field, which was about one-third of a mile from the yard, the cattle that had been put in the yard were most all lying down. I went among them and noticed one that did not seem right. I went to him and scared him up, and it was all he could possibly do to get up; he was just taken. When he got up he went staggering off, stepping high. His limbs seemed to be numb, and his sight affected. I drove him around, and gave him a small dose of saltpetre. The more I drove him the better he seemed to be. He improved until he walked all right, but seemed to be in great misery, and stood up and grunted until dark, when he lay down, and died about ten P.M. This, too, was a large two-year-old steer. *Now these were three of the very best of my cattle; and my water theory all gone up.*

"I took them out of this field and turned them into another. They cleaned it up, not any dying. Then I turned them into another which they cleaned up without any trouble, when I turned them back into the forty-acre field, in February, to clean it up, thinking the winter had by this time got away with the trouble. They ran there two or three days and one died. I then took them out of that field and kept them out, and made up my mind that I had just as well feed strychnine as leave them there."

Can more strong and positive practical testimony be given of the utter fallacy of the "dry murrain, short-of-water-and-salt hypothesis?"

Upon the same subject a Mr. W. E. Thorne, of Bladen, Webster county, Neb., writes under date of March 25, 1889:

"I have lost five head of cattle from running in corn stalks this winter—two on the 26th of December, the others at various times since. The last one affected was about two weeks since; but it recovered."

As to the salt and water business, he says: "*I took especial care to salt and water my cattle, and know that there was no lack of either.*

"After death I opened and examined the animals, but could not satisfy myself as to the cause of their death. *The first that died had no impaction of the third stomach whatever,* but in one or two of the later ones the contents were pretty dry and hard. When first taken they were very sick, and I thought there must be some substance in the stalks of a poisonous nature. The disease has been quite prevalent in various places about here, some fifty to seventy-five head having died within a radius of six to eight miles of this place."

ARE HORSES ALSO SUSCEPTIBLE TO INFECTION?

Upon this subject I am in receipt of the following letter:

"BYRON, NEB., March 18, 1889.

"MY DEAR SIR: About the 10th of December, 1888, my brother and myself fenced off about 200 acres of stalks and turned into them a lot of colts, leaving them there but a short time each day; the stock did well until about the first of January, when we lost seven colts within the same number of days; they appeared to become crazy and blind, most of them falling dead while running. We lost a great many colts in this way in Thayer county, Nebraska, and Republic county, Kansas. Yours, JOHN A. FISHER."

Was it the disease of which we are treating?

That there is a very direct connection between ensilage and a disease in horses has been known for a long time, though it is rare. The following quotation is interesting in this connection:

"WAS IT SILAGE OR SMUT THAT KILLED THE HORSES?

To the Farmers' Review, Chicago, Ill., March, 1889: The subject of feeding silage to horses that you have made so interesting in late numbers of the *Review*, is one of great interest, and more so as relates to the death of some horses said to have died from the effects of the silage. The case from Kentucky, I think, will bear a little closer investigation. A private letter received by myself from a party in Kentucky who had lost twelve horses from eating silage, said the surgeons had pronounced it cerebro-spinal meningitis, from silage eating. A noted veterinarian tells me that this disease is so rare that it would be impossible to have twelve or more cases closely associated; and gave it as his opinion that this silage was made from corn heavily charged with smut, (ergot;) and the conditions of the silage making were such that the formation of smut spores went on, permeating the entire mass; and by feeding the horses "all they could eat," they were killed by paralysis of the spinal chord. That might easily be mistaken for meningitis. What makes me think this is so,

is from a statement in this letter to the effect that the silage was badly spoiled along the sides, and occasionally layers would be found badly damaged. This would show that there was an entry of air from some source that would cause the decay, and the smut spores, taking advantage of this, would propagate, where the weather is no colder than it has been near Louisville, Ky., this winter. Hereabouts horsemen feed horses, brood mares, and colts large amounts of silage, "not all they will eat," but reasonable rations, and not a case have I heard of unfavorably. But in silo filling, stalks that are smut-laden are pitched "overboard," not put in the silo, and smut balls are twisted off from less-producing ones. The idea is to have good silage go into the pits. Now possibly this smut business may not have any existence in fact, so far as it relates to this Kentucky silo, and this guess about it may be all wrong. By the way, this gentleman who writes me from Crescent Hill, Ky., about this matter, instances a neighbor of his, twelve miles distant, who has a silo, and has lost a similar number of horses from the effect of silage, and remarks that "silage is killed in that section." I am pleased to see Prof. Henry's remarks in connection with this matter, for what he says is authority, and has great weight. His idea that a little acid in the silage makes it more favorable for digestion, recalls some experiments East this winter, when a certain amount of cider vinegar was mixed with the dry feed, and with the most beneficial and paying results. At the last silage congress in Cleveland, Dr. Ashmun, the health officer of Cleveland, testified that there was no danger or damage in a reasonable amount of acid in silage; and Dr. Stewart affirmed that 'without a certain per cent of lactic and acetic acid, there could be no digestion.' JOHN GOULD."

I have personally seen quite a number of cases exactly corresponding to the above, where horses were fed on corn fodder from a silo. This was some years ago, in Massachusetts; but I had no conveniences at the time for properly investigating them. Other cases have also been reported in different parts of the country; but no mention is made whether cattle were fed the same material or not, or whether any died therefrom. A gentleman of much experience also tells me that corn fodder will affect sheep and goats, but not hogs. That hogs will not become infected is well known. All Mr. Delaney's hogs were among the cattle all the time, and also in another outbreak to which I shall soon refer; and I have endeavored to kill them by the subcutaneous injection of such extreme doses as 6 fl. ccms. of a very malignant pure culture, but no signs of illness were shown.

PERSONAL EXPERIENCES WITH THE CORN-STALK DISEASE.

I much regret to have to say that I have never been able to see a single case of this disease as it occurs under natural conditions, my engagements in the laboratory, having no assistance, being of such a nature, and the outbreaks occurring at such a distance from Lincoln, that it has been impossible for me to make either clinical or necroscopical examinations: hence my notes on this very important subject must of necessity be most meagre and unsatisfactory, but to no one so much as to myself.

The first case with which I had any connection was of a most unique and unexplainable character, and remained a complete mystery to me until my investigations were completed on the Delaney outbreak at Fremont this year. It still remains mysterious in many ways, but it was from this case that I first obtained the etiological organism, and it is only from comparison of my notes of its method of development in and on different media that I am enabled recognize it as the same disease which occurred at Fremont a little over a year subsequently.

A SINGULAR CASE OF THE CORN-STALK DISEASE AT AMES, NEB.

On January 6, 1888, Dr. W. A. Thomas, a veterinary surgeon in the employ of the then Live-Stock Commission, as Inspector, came into the laboratory with two bottles, one containing the blood and the other some pieces of the organs of a steer which had died very suddenly at the feeding station of the Standard Cattle Company, at Ames, where he had been sent to inspect a number of horses on account of glanders. As to the steer, all he could say was that "it was considered well the night before—at least its condition did not attract any attention," and that it was found dead in the stable the next morning.

Knowing my interest in all such cases, Dr. Thomas hastily opened the animal and brought me the material mentioned, only reporting "the blood to have been fluid and of a dark color, the liver swollen, anæmic, and of a dirty yellowish-gray-brown color; the spleen much swollen, and weighing five pounds, and the kidneys swollen; cortical substance gray-red and anæmic, while the medullary was bright red."

I at once made a microscopic examination of the blood by covering-glass specimens, and cannot express the astonishment and perplexity which came upon me on seeing an apparently pure condition

of innumerable micro-organisms having the polar staining and belted appearance, as well as the size and form, of those I had already discovered in the southern cattle plague, and which I had demonstrated, beyond the possibility of question, to be the specific cause of that disease. These phenomena of resemblance were still more strengthened by the fact that, like the etiological organism of the southern cattle plague, this germ colored much more sharply in a fuchsin solution than in either a blue or a violet, which fact is an essential point of differential value between these two organisms and that of the true swine-plague, which colors better in the blue or violet tinctions than in fuchsin.

That the disease could not possibly be the southern cattle plague seemed to be shown by the fact that all and every experience in the latter disease contradicted its appearance in our northern climate in the midst of the winters. Again, I soon found that pure cultures of this new germ were rapidly fatal to rabbits, while I have thus far found these rodents immune to the action of the etiological organism of the southern cattle plague. I also found essential points of differentiation in the development of the two organisms on solid media, which have already been briefly noted in the previous article on the southern cattle plague, and which will find a more detailed description later on.

The wherefore of the death of this steer in so singular a manner, and among over a thousand others, all of which were presumably fed in the same way and with the same materials, was then a great mystery; and although we now know how it must have occurred, we still cannot understand why the outbreak was limited to a single animal, unless it happened to eat all the infested stalks and leaves on the place, (possibly a single stalk and the leaves attached to it.)

Immediately upon discovering the close resemblance of this germ to that of the southern cattle plague, and before my cultures had developed or I had tested its malignancy on any animals, I wrote to Mr. R. M. Allen, manager for the cattle company named, making inquiries as to the possibility of the steer's having come in contact with any feed that could have in any way been in contact with material polluted by Texas cattle during the previous summer or fall months, and received the following in reply, which is inserted on account of its historical connection with these investigations:

"AMES, NEB., Jan. 11, 1888.

"DEAR SIR: I cannot see any possible chance of the infection of the steer from hay polluted by Texans. The steer was from the J. E. Boyd herd, on the Cheyenne river, in Wyoming territory, and I do not know of any Texas cattle going into that country the past summer. We have Texas cattle of our own here, but they have all passed a winter in Wyoming. There have been no hogs about the cattle in any way. I am sorry I failed to examine another steer that died in the lot; thought it was constipated, [a condition common in the corn-stalk disease.—B.] but I think it died from a different cause. We lose a number of steers that die suddenly, but in such cases we generally find a diseased liver, [the liver is badly diseased in the corn-stalk disease.—B.] and other appearances of excessively rich and concentrated feeding. We did not know the steer was sick until we found it dead. We had examined this lot particularly for sick cattle, having recently lost one out of the same pen."

Here, then, was a new disease discovered, and one belonging to the group of extra-organismal (etiologically) septicæmiæ, and caused by still another member of the ovoid belted class of bacilli, or germs; but where the organism came from, or how the disease originated, was still a perfect mystery, which was not much cleared up by the appearance of a second outbreak in an entirely different part of the State, from which I also received material and the following communications:

THE CORN-STALK DISEASE AT CORTLAND, NEB., MARCH 1888.

The material from this outbreak came to me in a still more unsatisfactory manner than that previously mentioned. It seems it was originally sent to the State Live-Stock Commission, but as neither they nor the veterinarian were able to make any use of it, it was sent to this laboratory; and had it not had the name of "Dr. W. S. Brayton, Beatrice," on the wrapper, it would have been impossible to have traced the matter any further.

Microscopical examination of the fresh organs revealed the presence of, apparently, the same organism as had been found in the Ames case, and the inoculation of small animals enabled me to obtain pure cultures by which its identity with that germ was sufficiently demonstrated.

Upon writing to Dr. Brayton, I was favored with the following polite reply:

"BEATRICE, NEB., March 20, 1888.

"*F. S. Billings, Lincoln, Neb.:* DEAR SIR—Yours of March 18th received. The history of the cattle, as near as I can find out, is as follows: They were shipped from Osage, Iowa, about September 1, 1887, to Cortland, Neb. When starting from Osage, they were in apparent good health, and at Cortland were unloaded and given into the charge of a Mr. —— to winter. The cattle were herded on high ground, getting their water from a small creek which runs through the same. As soon as cold weather commenced, they were taken about five or six miles from Cortland and put into a lot containing somewhere from five to six acres, on a creek bottom, and were allowed to run to flax straw and oat straw for feed, and getting their water from the creek which ran through the lot—I think the same one that runs through the pasture in which they were herded during the fall, and in which they are at present. In this lot was some timber, (I do not know how much,) which was their only protection from the weather.

"From this lot they were moved to their present location, and allowed to run to a millet stack and to some oat straw stacks. Upon March 13th I held post-mortems upon two dead animals, one a cow, (in calf,) and a yearling steer. The cow had died the night before, and was in fair condition as regards flesh; but the steer was poor. I found upon post-mortem: First, that the blood was of an unnatural color, and seemed to be thick. The heart had a blood-clot in each side, and the posterior aorta contained a clot for about a foot from the heart. I found no lesions in the mouth; but when the stomach was reached the lining membrane of the rumen would peel off in large patches. The discharge from the bowels was a little thinner than natural, and streaked with blood. The lungs seemed in a healthy condition. The piece of lung sent was from the lung on the under side, which I think causes it to be so congested. The liver was about its natural size. The spleen about natural, with the exception that it looked blacker than usual.

"The history is as follows: These cattle would be all right at night, but in the morning there would be one or two that could not get up, but showed no signs of pain. Some of these would get up with help for once or twice, and then die in the course of three or four days. Others would not get up at all, and die in from twelve to twenty-four hours. The trouble seems to affect the cattle in the best condition. Cows abort their calves and seem to do well after it. There are no external appearances of disease. These cattle have had no shelter this winter except the timber already spoken of.

"I saw both hogs and horses among the cattle, but was informed that there had been none of them sick. I saw some of the hogs eating a portion of a carcass of one of the cows."

Though I had again discovered the same micro-etiological organism as I found in the material from the steer of the Standard Cattle Company, at Ames, Neb., a few weeks previously, and proved its malignancy in this case also, still I was completely in the dark as to its source or origin—that is, in what manner the cattle obtained it. That the disease was due to feeding on food polluted with the germ, I felt convinced. I had no data up to this time pointing to corn fodder as the cause. In fact, I doubted that whole business, though really knowing nothing about it, save that it seemed improbable that dry fodder alone, or even smut, could possibly cause it. It will be seen that Dr. Brayton does not mention corn fodder or stalk fields in his letter, and at the time I did not know enough to be suspicious and ask any questions in that direction; but he does mention millet and hay fodder, which opens up the question, can, or does, this germ invade these materials also? An answer can only be given by practical experience and observation with the assistance of the scientific botanist, and the pathological investigator.

In order to show the value of scientific investigation, even when the ultimate result sought for has not been attained, let me say that all these experiences clearly taught that the specific cause was in some way connected with the food the cattle were getting; so I advised Dr. Brayton to change the same entirely, which was done with the most happy results.

CORN-STALK DISEASE AT FREMONT, NEB., FEBRUARY, 1889.

By referring to an earlier part of this report the reader will see that:

"Dr. Osborn, State Veterinarian" (of Nebraska) "visited this outbreak, and on February 7th, said: 'There is no contagious disease known to veterinarians which affects the third stomach of a cow, and that was the seat of the disease in every case that I examined. The third stomach, or manifold, was packed with dry food which, taken into the fingers, crumbled like flour.'"

The above shows a terrible lack of necroscopic ability, for in the first place, the condition of the stomach named is, in a varying degree, common to every acute infectious disease in cattle, accompanied by an excessive rise of temperature; and again, as will be shown, there were essential, specific, and pathognomonic lesions in these ani-

mals of just such a disease, which the veriest tyro should not have allowed to escape his notice.

No sooner did I see the reports of this outbreak in the daily papers than the suspicion arose that it was probably the same disease from which I had previously examined material and procured the same etiological organism from Ames and Cortland. Unfortunately a most peremptory engagement rendered a personal inspection of the outbreak entirely out of the question. Hence, I dispatched the most trustworthy veterinarian at my command, to visit the outbreak and bring me such material (which he knew how to collect in a suitable manner) in sterilized bottles, with which he was supplied. As said above, I could not expect any detailed account of the gross pathological lesions; still Dr. Thomas's very brief report of what he did see is sufficient to show that much more serious lesions were present than those reported by Dr. Osborn — lesions which directly point to a malignant infectious disease that interfered most seriously with the circulation, and which must of necessity have been accompanied by an excessive rise in temperature. The animal examined by Dr. Thomas was killed by him and immediately opened, the material at once being placed in the bottles previously mentioned.

His report is as follows:

"SYMPTOMS AND POST-MORTEM CONDITIONS OF MR. JOHN DELANEY'S CATTLE; ALSO, HOW THEY HAVE BEEN CARED FOR SINCE DECEMBER 1, 1888.

"Lost fifty-two head up to February 7, 1889. Commenced dying five or six weeks previously.

"Mr. Delaney's herd was composed of 170 head, divided into three lots, viz.: one hundred cows and heifers, forty-six last year's calves, and twenty-four fat steers.

"Deaths have occurred as follows: seven fat steers, eight or nine calves, and thirty-seven cows and heifers.

"Mr. Delaney's herd is in good condition, though a few are thin in flesh. The one hundred head have been fed hay, and ran in the stalk fields during the day. The fat steers have been fed corn and hay. The calves were fed millet until February 4th. They also had some corn. The entire herd drank from one tank, supplied by a windmill, and all had a certain amount of salt.

"The hay fed is in very good condition. The cows also drank from the Elkhorn river when running in the field.

"The first symptoms noticed are switching the tail, some of them

shivering, followed by bellowing, staring eyes, chasing pigs and chickens — in fact, almost anything that will come in their way — straining so violently that many of them evert the rectum and evacuate only a small amount of fæces, somewhat covered with mucus. They become lame, paralyzed, and usually stand until a short time before death. The strongest ones live longest, and the wildest die in the shortest time. They are sick from eight hours to seven days. The majority of them become wild or delirious, and die within twenty-four hours after the first symptoms are noticed. Fifteen to twenty were shot to prevent them doing damage, and five or six did not get delirious.

"*Autopsy.*—Pleuro-pneumonia sufficient to kill, the entire lungs being congested, and the lower portions of the lobes solid; inflammation of the pleura, and about two gallons of serum in the thoracic cavity; stomachs all in good condition; liver very firm, and pale colored; gall-bladder well filled; urinary bladder filled; intestines inflamed.

"Mr. Delaney stated that in those he examined the liver looked 'half-cooked, or white, and the gall-bladder as large as a hog's bladder blown up.' Others stated that of those examined the majority of the gall-bladders were ruptured. In one case the rectum was divided, black, and gangrenous; also a portion of the small intestine the same.

"Mr. Delaney's farm is on the Elkhorn and Platte river bottoms, about five or six miles northeast of Fremont, a short distance from the Elkhorn river."

It will now be remembered that Dr. Osborn also said, "The hay and stalks are excessively dry, and the cattle not having sufficient quantities of water and salt, congests are found."

Just what "congests" means pathologically, is more than I can explain. Dr. Thomas was especially directed to look up the water question, and it is to be seen that in no way could the cattle have been without a sufficient supply of water to answer the requirements of nature.

The material brought in by Dr. Thomas consisted of fresh blood from the heart, which was coagulated, the coagulum being solid and of a dark purple-red color; serum over it straw-colored and clear.

There were pieces of the organs in a tin box that were somewhat frozen, for according to my orders they were to be allowed to freeze, and kept so, immediately after being removed from the animal.

Lung.—The pleura covering the piece of lung was much thickened, presenting an irregular, shreddy surface of a yellowish-red color, inter-

rupted by numerous small red centers. The vessels of the interlobular spaces were engorged, much resembling the condition seen in acute pneumonia in swine plague. [See Plate I.] The lobuli were solidified, some being of a grayish-red color, others purplish-red, while still others were of a yellowish-gray-red color and very anæmic. The cut surface was excessively œdematous, and the interlobular tissue was swollen. Many lobuli presented centers of a diffuse dark purple-red color, between which were others of a pearly-gray color. Others were dull gray, and still others yellowish-gray, with a varying amount of reddish tissue between them. Bronchial tubes filled with a straw-colored, coagulated material. As mentioned previously, such a description would answer equally well for a form of pneumonia met with in swine plague, especially in eight or ten-day cases. In fact, the structure of the lungs has such a close resemblance in cattle and swine that there is no pathologist living who could have told this piece of lung from that taken from a similar case of swine plague. This conviction would have been still more strengthened by the examination of covering-glass specimens of the tissues and the blood from this animal, but the fact that swine are known to be insusceptible to this disease entirely shuts out that probability. The closeness of the microscopical resemblance of this organism to that of swine plague is very well illustrated by the accompanying letter from Prof. T. J. Burrill, the most accomplished mycologist in this country, to whom I sent cultures and a slide:

"CHAMPAIGN, ILL, March 9, 1889.

"MY DEAR DOCTOR: Yours of the 3d instant reached me yesterday, apparently after some delay en route, and the box came this morning, safe and in good order. The tubes are all fertile, and as far as examined, have pure cultures. *I have not fully studied the microbe, but am not a little surprised that the thing is so near like hog-cholera in its microscopical characteristics.* But I find no difficulty in applying your description. I have already tried inoculation in a rabbit, and will further study your cultures and let you know result.

"I have not, but will also look up my old slides and compare. Will write you early next week. In the meantime, I congratulate you upon the progress already made in this entirely new work. Bravo!

"Hurriedly but truly yours, T. J. BURRILL."

To return to our pieces of organs:

Liver.—Capsule normal; cut surface very opaque; excessively swol-

len and anæmic, and of a dull greyish-brown-red color; peripheral portions of the acini of yellowish-grey color; center reddish.

Kidneys.— Cortex much swollen, anæmic, opaque, yellowish-grey-red in color; vasa and tubuli recti much distended; medullary substance bright red, interrupted by a very pregnant distension of the tubes and an occasional large blood-vessel. A small piece of the small intestine presented a very much swollen mucosa, covered with a thick viscid coating, and of a diffuse yellowish-red color.

Covering-glass specimens from all these organs gave apparently pure representations of one and the same organism, which corresponded exactly to those found in the Ames and Cortland outbreaks. A hanging drop, prepared directly from the blood coagulum, at once shows the organism to possess most active movements, corresponding exactly to those of the swine plague and southern cattle plague, and possessing the same manner of development, so that, in order to save needless repetition, the reader is at once referred to the preceding treatise upon the southern cattle plague for a description of the morphology and biological phenomena presented by this corn-disease organism during its course of development.

HISTIOLOGICAL EXAMINATION OF ORGANS FROM FREMONT COW.

As is mentioned in another part of this paper, only a small part of the diseased organs from the cow were brought to the laboratory. This examination has, therefore, reference to sections made only from such.

Lungs.—Under low power a section from the lung presents numerous centers of consolidation, the outlines of which are more or less sharply defined from the surrounding tissue, these centers having irregular shapes and extent, the cellular products filling the alvioli being much more compact and dense in some than in others; but what is most striking is that the most intensely infiltrated alvioli are generally those most distantly situated from the bronchiole from which the irritation to the parenchymatous tissue extended. The balance of such a section presents alvioli, the majority of which are filled with a slightly granulous, very delicate, slightly yellowish-gray material, in which are to be seen numerous nuclei, the same being much more numerously represented in some spots than in others.

The larger vessels are compactly filled with a similar material.

The outlines of each alvioli are distinctly seen, and marked by nuclei-looking bodies along their course. The mucosa of the bronchioles in these sections is swollen and covered with a considerable amount of a yellowish-gray material. In many alvioli the yellowish-gray material spoken of is more or less mixed up with nucleated material. Upon closer examination with a higher power, the aforesaid yellowish-gray material in the alvioli is in many cases seen to be of an almost homogeneous character, a few delicate thread-like lines only running through it here and there. An occasional nucleated body is also to be seen. In others, on the contrary, the mass is seen to be composed of small, round cells, (red blood cells,) their contour being more or less distinctly marked, each cell being very clearly straw-colored, transparent, and refracting. Among these cells are to be seen numerous thread-like fibres extending in different directions, as well as quite a number of round cells, mono- and multo-nucleated, the former of which are in general much larger than the latter. The vessels taking their course along the septa of the alvioli, are distended and marked by the presence of numerous round cells with distinct nuclei in close apposition with the walls, many of which are collected along the outside of the vessel in more or less extensive masses. The contents of the consolidated alvioli is made up of the usual-formed round cells. The interstitial spaces appear to be completely filled up with round cell infiltration.

Liver.—Offers nothing essentially striking, other than the changes upon the cells common to acute parenchymatous hepatitis. The cells are very much enlarged, many of them being but a diffuse granulous mass, presenting nothing of a nucleated character. In others the nucleus is quite pale, the nucliolus not being at all visible, while in others again, both are distinctly marked. Both around the larger and smaller blood vessels decided lencocytic migrations are seen to have taken place. In several places are circumscribed accumulations of round cells more or less toward the center of an acinus, and lying in proximity with and in apposition with one side of a central vein, while others are more or less in the body of the acinus; but a very close examination will show that they surround or extend from a capillary.

Kidney.—A section of the kidney shows all the changes of acute parenchymatous nephritis. The urinary tubes are swollen, the contents

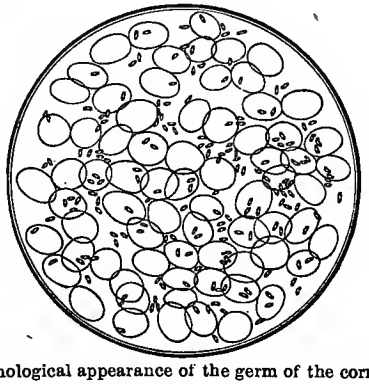
of most of them being nothing but a granulous detritus. Cellular structure absent. In other tubes sometimes one swollen cell with defined outlines and prominent nucleus may be seen attached to the membrana propria, while in others several such cells may be present, but seldom in continuity. But a very few tubes, and then for but very short distances, present normal characteristics. Here and there, more or less circumscribed, but dense, round-cell infiltration may be seen to have taken place at the expense of the parenchymatous tissue, which self-evidently antedated the acute changes in the other. Along the line of the capillaries a limited round-cell migration has occurred, especially in points where the vessels are distended owing to compression of their lumen in the immediate vicinity, by the intensely swollen parenchyma, which led to local interference with the circulation.

EXAMINATION OF THE ORGANS FOR MICRO-ORGANISMS.

The organism described in these pages as the etiological moment in the corn-stalk disease, is scattered profusely through sections of the liver, being mostly situated intra cellular, that is, between the cells of the parenchyma, and plentifully represented in the blood vessels, as well as having been taken possession of in quite large numbers by an occasional round cell. In no place can they be seen to have formed threads, as is the case with its near relative, the germ of swine plague, more especially in the kidney. Occasionally two or three individuals can be seen linked together.

The above description will answer for the dispersion of the organism in the kidneys, but it may as well be mentioned that they are numerously present in the detritus of the urinary tubes; when the section has appropriately met, the object can be seen in great numbers within the capillary loops of the Malpighian tufts. The swollen vessels between the capillaries are more or less filled with them. They are also very profusely represented in the lymph glands under the same conditions, but not so much so in the lungs, which, to my mind, is singular, for as has been noticed in those of the Fremont cow, the alvioli were largely filled with blood or extravasata of a more or less hemorrhagic character; but it may have been that there had been no rupture of the walls of the vessels, and that the red cells passed out by diapedesis only, and that the micro-organisms could not, under such

circumstances. That infection in this disease is unequivocally *via* the digestive tract, has been already pointed out, and hence their presence in the lungs from the respiratory tract is absolutely excluded. I must say that I was not surprised at finding so few germs in the lungs under such circumstances.



Mature morphological appearance of the germ of the corn-stalk disease.

ESSENTIAL CHARACTERISTICS OF THE DEVELOPMENT OF THE CORN-STALK DISEASE ORGANISM, AND THE POINTS OF DIFFERENTIATION BETWEEN IT AND THOSE OF SWINE PLAGUE AND THE SOUTHERN CATTLE PLAGUE IN AND UPON DIFFERENT CULTIVATING MEDIA.

The germ grows well and more characteristically at ordinary room temperature than in thermostat.

Potatoes.—Upon the cut surface of sterilized and steamed potatoes, the germ of the corn-stalk disease grows as grayish-white, somewhat elevated colonies, while that of swine-plague develops in a sort of yellowish, dirty-olive color, sometimes resembling muddy coffee, and that of the southern cattle plague in primarily straw-yellow color, which eventually assumes a slightly reddish shade. Very soft moist potatoes will affect the growth and color. [See plate II.]

White of Eggs.—The most practical way of sterilizing this material, and of getting it in a convenient form, is to steam the eggs until cooked hard, and then remove the requisite portion of the shell with sterilized forceps, after which they are placed in the moist chamber, prepared in the same manner as for potatoes or plate cultures.

Upon this medium the corn-stalk germ grows as clear yellow colo-

nies, with slightly raised edges and is to be easily distinguished from that of swine plague, which develops as a somewhat oval projecting semi-fluid mass, the center being the much more prominent, and from that of the southern cattle plague, which presents a buff-colored colony. [See plate III.]

Agar-Agar. (Plain, not glycerine.)—Upon this medium, when the surface is dry and not accidentally moistened by the fluid in the bottom of the tube, the corn-stalk germ develops in a really characteristic manner in contradistinction to the other two. In the color of the development there is nothing essentially different to be seen; but in comparison with the swine-plague organism the edges of the cultures are more distinctly scolloped, the separating lines extending deeper into the body of the cultures. But a marked point of difference is, that commencing from the line of inoculation, the corn-stalk germ forms lines, or rays, which are finally limited by a diffuse semi-transparent border. Each of these lines is made up of individual colonies. Again, the cultures are much more dry, and less viscid, than those of swine plague, and adheres much more closely to the underlying agar; in fact, when old it will break off in fragments on attempts at removal with the wire. As said, in order to get these effects, the agar must be just right, and have a dry surface. On agar there are no essential points of difference between the swine plague and southern cattle plague organisms except rapidity of development and a slight variance in the irregularity of the edges of the cultures. [See plate IV.]

In and on Beef-Infusion Gelatine. (Punctures.)—The growth in the gelatine itself, of these organisms, offers nothing of a differential characteristic nature; but their development upon the surface varies considerably, the germ of the corn-stalk disease developing and spreading over the surface the most rapidly and with more leaf-like extensions. It is also more dry and lusterless than either of the others. That of the southern cattle plague is next in rapidity of development, while that of the swine plague spreads more slowly, but in a thicker mass, and with more prominent edges. Both this and that of the southern cattle plague form a moist and more or less viscid colony, while the corn-stalk germ adheres to the gelatine, and is inclined to break up on removal with the wire, especially if the cultures are a little old. [See plate V.]

Upon the Oblique Surface of Beef-Infusion Gelatine.—As neither of these organisms cause this medium to become fluid, it was quite interesting to test them on gelatine prepared in this way. The germ of swine plague here develops or shows more of a yellowish-white shade than the others, and has a greater degree of opacity, the edges of the growth being very delicatety irregular. The corn germ extends the most rapidly, and the growth is more pellucid-pearly white than the others, the edges being much more deeply scolloped. Toward the bottom of the growth a delicate, smooth, pearly-white edge embraces the scollops. The southern cattle plague germ develops the most slowly of all, and is of a milky-white color, but less intense than that of the swine plague. The edges of the growth are more delicately irregular than in either of the others. [See plate VI.]

INOCULATION-EXPERIMENTS WITH PURE CULTURES OF THE CORN-STALK ORGANISM.

The next step was to prove the malignancy of the pure cultures thus obtained. To this end there were inoculated February 11, 1889:

1. One full-grown buck rabbit with a pure bouillon culture. In sub-cutis of right ear, two drops; in that of the inside of right flank, three divisions of a one-ccm. syringe. The ear was inoculated especially to see if there would any extreme degree of tumefaction follow.

2. One male full-grown Guinea pig. Three divisions of syringe in inside of right flank.

3. Mouse. One drop in same locality.

Morning of the 12th. Rabbit ill; will not move unless made to; not eating; no local tumefaction. Mouse very ill; coiled up in corner of cage, and only moved on disturbance. Guinea pig same, but not so much depressed; nibbling a little food.

At 3 P.M., 12th. Mouse just died. No œdemia at point of inoculation, but local vessels engorged. Mesenteric vessels engorged; and serosa of abdominal cavity swollen and glistening; muscles looked as if cooked; spleen, liver, and kidneys swollen, and very full of blood; lungs congested.

At 3:25 P.M., 12th. Rabbit dead. Skin and subcutaneous tissues at *locus-inoculationis* much engorged, but no œdemia present; of a diffuse red color, with marked injection of larger vessels. No effects in ear other than mentioned; no effusion in cavities of the body;

blood extremely thin and of a purple-red color; lymph gland much swollen and very juicy. *Liver* swollen; serosa normal, but the parenchymia reflecting through it was of a yellowish gray-red color; peripheries of acini yellowish; center red; cut surface very friable, opaque, and anæmic; yellowish gray in color. *Kidneys* swollen; capsule nonadherent; cortex anæmic, opaque; gray-red in color; medullary substance of a diffuse dark-red color. *Spleen* swollen; pulp very juicy and soft; vessels of mesenterium engorged; those on curvatures of stomach the same; mucosa of stomach swollen, covered with thick, viscid coating, diffuse bright red in color; contents of small intestines fluid; mucosa much swollen, yellowish red in color, and covered with a viscid coating; contents of large intestine pultaceous; mucosa not swollen; lungs engorged. Pure cultures of same germ as found in material from Fremont animal derived from each organ, and accurately tested by comparison with original cultures, as well as microscopically; same organisms in small intestines and isolated from the others present by plate cultures.

At 2 P.M., 13th. Guinea pig dead. Lesions same as in rabbit; cultures conformable.

These experiments demonstrated three facts of essential differential diagnostic value. Aside from those demonstrated by the cultivation experiments, these inoculations showed that the organisms in question could not be that of swine plague on account of its acute fatality. It could not be that of the southern cattle plague because of the immunity of rabbits to that germ in such small doses. It could not be the germ of the German "Wild-seuche" because of the total absence of enormous œdemia which invariably follows such inoculations in that disease.

It must then be a new disease!

EFFECTS OF INOCULATIONS WITH THE SAME MATERIAL IN A STEER AND HOG.

These inoculations were made on the 9th, two days previous to those in the small animals mentioned, six ccms. of a pure bouillon culture being injected under the cutis in each case. The hog was not affected at all, and with that we will leave it.

Steer calf five months old:

10th. Temperature 10 A.M., 39.50° C.

11th. Temperature 10 A.M., 39.50° C.

12th. Temperature 10 A.M., 39.70° C.

Locus inoculationis slightly swollen and hot; animal lying down and loth to move; respiratian 60 a minute; pulse much accelerated; right lung solidified for the lower half; augmented vessicular respiration, with some thickening of the sound in superior half of this and whole of left lung; drinking much, but eating little.

At 9 A.M., on the 13th, the temperature was 41° C; respiration 60; pulse as before; made to rise with difficulty; right lung still more solidified and some pleuritis in lower portion; eyes wild; conjunctivæ injected; yellowish material escaping from nostrils; severely constipated; fæces dark and hard; urine albuminous.

At 5 P.M. the temperature was 41.50°; manure becoming pultaceous; otherwise same.

At 9 A.M. on the 14th the temperature was 40.70°; condition about same; animal emaciating rapidly; discharge quite fluid, and of a yellowish color.

At 9 A.M. on the 15th the temperature was 40.70°; manure a little more solid; respirations somewhat easier; animal eating a little; lungs clearing up somewhat.

At noon on the 16th temperature was 39.90° C.; evening, 39° C.; animal improving.

At noon on the 17th temperature 36.2° C.; evening, 36.2°.

This looked like death. The animal was shivering, and terribly emaciated, and I felt sure of an autopsy in the morning; but on the—

Eighteenth—Morning temperature, 37° C.; evening, 37°.

Nineteenth—Morning temperature 37° C.; evening, 37.1°.

Twentieth—Morning temperature, 37.1° C.

The animal eventually recovered, though it became excessively emaciated. On the 16th I washed the inferior surface of its tail with Cor. Sub. 1:1000, and with sterilized knives opened to the local vein, allowing the blood to flow directly into a sterilzed homœopathic vial. From the same I again obtained pure cultures of the germ, which were proven by cultivation and comparisons with the various others, all of which were kept up on the different media mentioned until the investigations were terminated to my satisfaction.

A FEEDING EXPERIMENT WITH PURE CULTURES.

Fed a full-grown female rabbit with pure bouillon cultures from Fremont cow, by pouring 25 ccms. between the leaves of a quarter of the head of a small cabbage, two days in succession, beginning with February 14th.

There was no change on the 15th or 16th, the animal eating with avidity. On the 16th it was somewhat quiet, but still ate its rations pretty well; 17th, back arched, hair bristling, sitting quiet in corner of cage, respirations somewhat increased, eating but little; 18th, not eating at all, remained in one place, back much arched, respiration very rapid.

At 9:30 A.M. on the 19th it was dead in the cage on my arrival at the laboratory. It was somewhat emaciated; musculature very pale. Peritoneum swollen and diffuse pink-red, with occasional punctiform red spots scattered through it; serosa of stomach and small intestine swollen and clouded; vessels much engorged. Liver swollen, of a mottled grayish-yellow-and-red color; gall bladder distended, duct open into intestine; outside surface anæmic, opaque, and yellowish-gray-red in color; acini swollen; peripheries yellowish-gray in color; center reddish; spleen excessively swollen; pulp soft and juicy. Kidneys swollen; cortex opaque and anæmic, yellowish-gray in color; medulla diffuse red; thoracic cavity contained about a tablespoon of yellowish-red fluid; right lung attached to ribs in all directions and solid throughout; pericardical sack totally obliterated; portions of left lung solid, and red centers throughout solidified parts. Urine albuminous.

Cultures from heart, blood, spleen, liver, lungs, and kidneys, contained the same germ as in previous cases, and developed in same manner.

A FEEDING EXPERIMENT WITH CORN FODDER FROM FREMONT, NEBRASKA.

Immediately upon finding the same micro-organism in the blood and organs of the cow killed at Fremont that I had found in the same kind of material from Ames and Cortland the previous year, and feeling there might be a hidden truth behind the dry-corn-stalk-no-water theory, I wrote to Mr. Delaney, of Fremont, to send me some of his corn-stalks, and to strip off the leaves so as to save bulk. He

very fortunately misunderstood me, and sent me a small package of the stripped leaves, totally insufficient for any feeding experiment in cattle. On account of extreme demands on my time, I let the material remain in the package, and gave no thought to it, until February 26, 1889, when Dr. E. O. Shakespeare, of Philadelphia, being here, and wanting a piece of paper to wrap something in, I undid the corn-leaves and gave him the paper. As a mere matter of curiosity I gave the leaves to a rabbit to eat, cutting off all other feed, but giving it an abundance of fresh water twice daily. So little did I think that the leaves would lead to any positive effect in the rabbit, that I gave no attention to the animal, until my man told me it was dead, on the morning of March 4. In fact, it had just died as we got to the laboratory. It had been eating green food for the past three days, but had not touched that given it on the 3d. My interest in the matter was very great, and no time was lost in making examinations and cultures. In the blood, liver, spleen, kidneys, and mesenteric lymph glands, the same germ as that found in the cattle was present, so far as one could judge from a microscopical examination. Cultivation experiments gave all the points of differentiation from swine-plague, which experience has shown can be relied upon, The cultures were all strictly pure, as shown by cultivations on potatoes, eggs, and in gelatine.

The lesions in the rabbit were briefly these: Swelling of the peritoneum, with straw-colored effusion in cavity; liver excessively swollen, very pliable, and almost a phosphorous liver, in portions, in its degree of fatty degeneration; spleen, same as to swelling, and pulp almost semi-fluid; kidneys: cortex almost pure yellowish-gray, opaque, and anæmic, also friable; stomach two-thirds full, mucosa intensely swollen, and covered with a thick, viscid coating, beneath which the tissues were almost diffuse purple-red; contents of small intestine fluid, mucosa very thick and swollen, and covered with a glairing, viscid coating, and anæmic, as in feeding experiments with the swine-plague germ; large intestine, contents pultaceous, mucosa but little swollen, though much injected; mesenterial lymph-glands resembled the strawberry glands seen in hog cholera excessively swollen, vessels of membrane engorged; thoracic cavity, heart and muscles opaque and very anæmic and friable; bronchial lymph-glands swollen, diffusely-red, and juicy; left lung hyperæmic, right normal.

Thus it will be seen that I have followed this germ, first finding it in the cattle, and then tracing it from them to the corn, demonstrated its malignancy in that material, and, beyond that, the accidental sending to me of the leaves instead of the stalks by Mr. Delaney, and the positive result in the rabbit fed with those leaves, closely conforms to the practical fact that cattle when turned into such stalk fields naturally take to the leaves and tender top shoots, leaving the hard, dry stalks more or less untouched, and hence it is from them that they become infected. I believe this to be the first actual demonstration of a germ of a malignant nature infesting our grasses or grains during their development, and having disease-producing properties for certain forms of animal life when fed upon them. The question still remains open, so far as this germ is concerned, Does it also penetrate the ear? That it is in the stalks I myself have demonstrated by microscopic examination and cultures; but with this singular result: *It is not disease-producing when inoculated from this source in either rabbits or mice, while from the leaves it is, as we have seen from the feeding experiment in the black rabbit.*

This fact again opens up a still more interesting question, viz.:

Does the nature of the soil, (nutrition,) or the chemical properties in which these extra-organismal infectious elements primarily live, or into which they get, cause their specific infectiousness in relation to animal life?

We do know that the chlorophyll, and perhaps some other elements in this case, of the leaves of the corn, has a different chemical composition from the pith of the stalk, or even its woody covering, unless it be its extremely superficial layer; hence we must face the question —as this organism is not disease-producing when derived from the stalks—Does the germ cause a chemical decomposition in the elements and juices present in the leaves, or does the chlorophyll itself supply a material which causes such changes in the physiological attributes of the germs as make them disease-producing for a certain length of time? For it is well known that in our artificial media they either lose this infectious property in time, or can be made to do it at our pleasure. In fact, we are able to graduate that property at will. As to the swine-plague germ, I have experimentally demonstrated that a mitigated culture of these germs, which will not seriously affect a bunch of pigs when inoculated with one ccm. under the cutis, though

sufficient to protect them from the natural infection on the most severe and repeated exposures, still gets into the contents of the intestines of the inoculated animals, and when passed off in their manure, and remains there for a time under comformable conditions of temperature, becomes exceedingly virulent toward healthy hogs if put where such manure is, their food being scattered among it so that they are obliged to consume more or less of such infested manure as they seek their food.

Again, we have to discover whether this corn-germ also infests grasses and other fodders, such as millet, as is rather indefinitely indicated in the letter from Dr. Brayton regarding the outbreak at Cortland, Nebraska, to which reference has been made.

These are all questions of great hygienic importance to our stock-raisers, and perhaps to man as well; for should it be demonstrated that the ear of corn is dangerous also, we have to face the question, Is it in the grain also, and is the meal from such grain dangerous to humanity as food? For in these things we cannot always depend on cooking, though probably we can in this case, as this corn organism is not spore-bearing.

The pathological investigator cannot settle this corn-disease question altogether. Having traced it directly to the corn, and demonstrated the futility of "dry murrain," impacted third stomach, corn-smut, insufficient water and salt, and what not absurd theories as to the origin of this disease in cattle, it now remains for the botanist, or perhaps more truly, botanical mycologist, the "simon-pure" bacteriologist, to trace the organism in corn, and tell us, or better, the farmers, how it affects corn, and how they can distinguish such corn, (for the disease also has its language in corn,) so that the breeder will be enabled in the near future to avoid using such corn-fodder in feeding his cattle and horses. One thing is very certain—this corn disease is exceedingly local in its extension, being not only limited to certain fields, but even to certain portions of such fields, and most probably to certain stalks in a hill, or at least to individual groups of stalks.

That much of this important work has already been done may be seen by reading the following invaluable letters from my esteemed friend Prof. Burrill, from which it will be seen that that genial investigator should be accredited with the first real discovery of this interesting though unfortunately fatal organism to our live stock. But as Priscilla said to John Alden, "Prithee speak for thyself, John:"

"CHAMPAIGN, ILL., Feb. 4, 1889.

"MY DEAR DOCTOR: The disease you speak of in cattle is a common one in this vicinity in the fall and early winter when the animals are turned into corn-stalk fields, and has occurred, I think, under other conditions. The veterinarians have pronounced it due to an impaction of the third stomach, just as your men have. I have not myself seen a post-mortem. The thing is of much interest practically and scientifically, and I will look out for it.

"My best regards to you, and believe me, Very truly yours,

"T. J. BURRILL."

"CHAMPAIGN, ILL., March 11, 1889.

"MY DEAR DOCTOR: I find on comparison with my old slides that the microbe from your cultures differs mainly in its smaller size. I send you a slide taken from the diseased corn-stalks, (maize.) This was two years ago. I did not deem the matter sufficiently worked up to publish, but did publish the account of the organism in broom corn and sorghum — a different thing from this in maize. This corn-stalk trouble is, as I found it, very local, occurring in a given area of a field and not elsewhere. In several instances I found it only upon low spots, and in two instances on ground that had been poud holes until the year before, when they were tile-drained. In another case a whole field of ten acres on rather sandy soil, and in clover the year before, was affected. Last year a man sent me some green stalks which he said came from a forty acre lot, nearly all of which failed, though soil and season seemed favorable. The stalks showed plainly enough the special trouble of which I now write. The most marked effect is to be noted in the leaf sheaths, and if you can get a culture at all from the corn-stalks, I think you can best do it by stripping off some of these sheaths and taking material from the inner surface of the tissue in corroded spots. Your cultures all seem pure, and as I have now some young corn growing, I will try the effect of the same upon it. Your germ is motile in liquids, and in this agrees with my corn microbe; neither liquefies gelatine. But I did not sufficiently study the thing at the time to be able to compare much further.

"Yours, T. J. BURRILL."

Upon examining the slide sent to me by Prof. Burrill, I find the difference in size mainly due to the fact that he used a blue tincture to color his germs, which, with this organism, gives a diffuse, washy outline, thus making it look larger than when colored with fuchsin, when its outlines are very sharply marked. However, I have no doubts but that my testimony and that of Professor Burrill will eventually completely coalesce, though being an infinitely more skill-

ful bacteriologist than I pretend to be, being but an embryo pathologist, I do not doubt that he may not only find something to correct, or differ from, in my bacteriological work, but add much of interest and value to it, though not militating at all against its general correctness. My interest in bacteriology does not extend an iota beyond the relation of specific germs to the disease caused by them and such points as are directly essential to the differential diagnosis of one germ from another, but only as they bear upon disease. A bacteriologist I am not, and have no interest whatever in adventitious germs, or bacteriology *per se*.

HOW THE BURRILL-BILLINGS GERM AFFECTS CORN.

My worthy senior and co-laborer in this important field of research sends me the following as his special contribution to that part of the story which it is beyond my ability to write upon, at least at present:

"*Dr. F. S. Billings, Lincoln, Nebraska:* DEAR SIR—According to your request, I herewith send you some account of a disease affecting growing corn (maize) in the field. It must be premised, however, that the malady as affecting this staple crop has not been fully worked out, because for some unknown reason the attempts to communicate the disease to healthy plants by the application of culture-materials containing the living organism derived from diseased plants, have not been successful. I am thoroughly assured, however, that these failures are from some fault in the methods tried, rather than in the want of having the true disease 'germ,' for a specific organism is too uniformly present in the affected tissues to permit us to consider it accidentally connected therewith. Moreover, the progress of the disease from cell to cell corresponds exactly with the spread of the minute organism in these tissues. It is never found far from cells which plainly show the disease characteristics, though I have succeeded in making cultures from parts appearing healthy upon the very border of the affected portions. But this only goes to show that the healthy cells are invaded before they present the characteristics of the diseased cells; that is, the organism causes the disease, not the disease the organism. There was a time, perhaps, when this latter alternative might have been seriously considered, but if so, surely the time has gone by. While no one will rest content with the simple presence of a microbe even constantly in affected tissues as full and conclusive proof that such microbe is the actual active agent in the disease, still it must be admitted that this is exceedingly strong evidence of such active agency. In the present instance I have but little hesitation in saying, without successful inoculations, that the organism which has

been so often found in the diseased parts of the corn plant, and which has repeatedly been obtained in a state of purity by cultivation methods, is the direct cause of the mischief observed. I am the more confident of this from the fact that but few inoculation experiments under natural conditions have been tried. Of course continued failure of these last would shake the confidence now felt, but up to the time of this writing there is no cause for such skepticism. It should be said that although some work was done upon the disease during the two seasons of 1887 and 1888, other duties were permitted to crowd out this special investigation except as stated below.

"Further, it must be said that so far as I now know, the evidence that the organism found by me in diseased corn is identical with that sent to me by yourself in cultures from rabbits dead from eating suspected corn stalks, rests entirely upon their microscopical appearances. I had only mounted slides of the former for comparison, the cultures having been lost. Under the microscope they do seem to be identical. Both are actively motile, as my notes of the former examinations indicate for that direct from corn, and as is readily seen in the preparations of the living 'germs' from the rabbits. Neither, it appears, produce spores, at least endospores. They behave the same in the staining characteristics so far as tried by me. Now for the notes you request.

"The disease in the growing corn may commence at any time during the warm season. According to my observations, it is most likely to become noticeably apparent after midsummer, or after the corn 'shoots,' though this is by no means always the case. Very often it occurs only upon certain pretty clearly-marked areas or patches in the fields. I think this must be very generally the case, for only two exceptions have fallen under my observation. One of these exceptions was a field of about forty acres, the other of less size; both had fertile soil and were well cared for. Neither paid the expenses of cultivation. As generally found, the affected patches are easily recognized. The corn fails to grow as in the healthy areas. Its stunted size at once arrests the attention. Diseased stalks may, indeed, be found as large as the largest, but it is probable that these became later affected. In one instance, an area of about an acre, planted with the rest, did finely until the young corn was about six to eight inches high, after which it died so completely that the farmer replanted the patch. This latter planting did well so far as is known.

"Along with the dwarfed appearance designated as common, the lower leaves prematurely die, passing through the stages of becoming yellowish-green, then yellow, then withering away. Upon closer examination it will usually be seen that there are certain spots, more especially upon the basal part of the leaf, which is wrapped closely around the stalk, having a different discoloration. These are brown

—watery-looking at first, then darker, and finally dead. Occasionally there are livid red spots or patches in the same situation, but I am not sure that these mean the same thing. These specially affected spots vary in size from mere points to those of several inches across, often longer, in the direction of the veins of the leaf or leaf-sheath. It is in such diseased parts that the microscopic organism believed to cause the trouble can always be easily found, and from which cultures are readily made in beef broth, the juice of the corn stalk, etc.

"The remaining characteristic of the disease is really the one most conspicuous to those who search carefully. When the corn suffers worst from the malady in question, the roots are badly affected. Beginning with the oldest and lowest, they die and decay in the ground. At length the stalk is held upright only by the later-developed 'brace' roots, and even these may slowly corrode away. Under such circumstances the affected stalks are very easily pushed over or pulled up from the little hold they have in the ground. If the plant is carefully dug and the affected roots examined even with a magnifier, no evidence can be found of the work of worms or of insects of any kind. The roots simply die, though no wounds are to be found. The external layer rots most easily and quickly, and the woody inner part may then be pulled out like a string.

Of course when the roots are thus affected the whole plant suffers, ceases growth, fails to mature its ear; anyone and everyone who observes at all knows that something is the matter. If one now looks closely at the brown spots on the leaf-sheaths or roots during the first stage of disease, he will often find little collections of gelatinous-like exudation. Crush a minute bit of this under a microscopical cover-glass and examine with a high power of compound microscope, and the living organism to which we ascribe the disease can be seen in innumerable numbers.

So far as observed, corn on rich lands is more likely to suffer; not unfrequently that in low places recently broken up from the sod of wild grasses seems to be most affected. Sometimes instead of distinct areas in a field being alone injured, scattered stalks throughout the plantation are diseased; but this seems much less common.

"The corn disease as now described is a very similar disease to that affecting broom-corn and sorghum, but is nevertheless due to a distinct organism, having at least some differences in mode of development and action under different circumstances. The broom-corn disease was deemed sufficiently understood for publication in 1887. The topic was presented by me to the meeting at Cleveland, Ohio, of the Society for the Promotion of Agricultural Science, and the paper was published in the proceedings for that year. It was also published in the transactions of the American Society of Microscopists for the same year. I am truly yours, T. J. BURRILL."

"*Champaign, Ill., May 1, 1889.*"

NATURE OF THE CORN-FODDER DISEASE.

It is not necessary to go into a very detailed discussion of this point, as it has been sufficiently treated in the preceding article upon the southern cattle plague, (Part III,) and even more fully in my report upon the swine plague of the United States.

Like both of these pests, which so seriously interfere with the prosperity of the live-stock interests of the United States, this "corn-fodder" disease has nothing of a contagious character about it. It does not owe its primary origin to the presence of a diseased animal in the first place, or of any material from such a diseased animal, among healthy stock; it is not an endogenous (generating from within) disease. On the contrary, a better example of an exogenous (owing its genesis or origin to external circumstances) disease could not possibly be found. It most aptly illustrates my previous endeavors to impress upon the medical world the absurd folly of the continued use of the words contagious and infectious in any endeavor to express class in disease. The word "contagious" simply expresses to us the fact that the infection of a healthy individual in a given disease either took or can take place by means of contact either with an individual diseased with a specific disease, or with some material directly from such an individual. Correctly speaking then, the word contagious only means that the primary origin of the inficiens, (infecting material,) was within the body of an already diseased animal. Any other definition of this word is absurd; but the terms which I have selected to express this class of diseases, while equally as technical as that of Pettenkofer, "endogenosis," is more self-evident to the ordinary class of readers, viz: "*intra organismal;*" that is, originating within or from a diseased animal organism. On the contrary, I have called the opposite class "*extra-organismal diseases,*" simply because their primary origin must be invariably sought in the external surrouudings or conditions. In the disease in question we have clearly demonstrated that its primary cause is to be sought in the corn fodder, and perhaps the grasses, which certain susceptible animals eat.

Again, this disease bears a very close relation to both the southern cattle plague and the swine plague, in that it is an absolute local disease. Mr. McKelvie's letters demonstrate that fact in *optima forma*, for he tells us that it was the corn fodder in a certain field which was dangerous to his cattle; and all practical experience in every part of

this country serves only to confirm that statement, without even a single contradiction. Professor Burrill tells us the same thing with regard to the disease in the corn itself, and I feel very sure that more exact study will eventually demonstrate that in many cases this localization, with reference to disease in corn, will be found so centralized that even a single stalk in a single hill, or a group of stalks, will alone be found diseased, while in other cases there will be several, and in still other places whole groups of stalks, or even portions of a field or the major part of it, will be complicated as witnessed in the "forty-acre" field quoted by Prof. Burrill in his second letter.*

THE CORN-FODDER DISEASE A SEPTICÆMIA.

Were I to be asked what disease of animal life this corn-fodder disease most closely resembles, I should say it is the exact counterpart of the genuine swine plague, and, in fact, most respectfully refer the reader to page 320 of that report upon the "intra-vital phenomena" in that disease. In the corn-fodder disease in cattle, every organic lesion and every variety of lesion seen in swine plague will be seen, with the exception of the ulcerative and neoplastic lesions, and perhaps diphtheritic, seen in the intestines, the two former of which are primarily due to idiosyncrasies of structure in the hog, and only secondarily to the action of the bacteria causing that disease. Here, too, we have the same excessive parenchymatous changes in the great glandular organs of the body common to all acute diseases of this character; here, too, we have pneumonia in all the various types seen in swine plague, and again, one form bearing the very closest resemblance to those in swine plague, marked by the engorgement of the interlobular vessels and coagulation of the blood within them, which I illustrated in plate XIII. of my swine-plague report, (plate I. of this,) and which is equally well applicable to this corn-fodder disease. In both species of animals the pregnancy of this lesion is due to the peculiar loose and open character of the interlobular tissue and the large vessels circulating therein. Again, in both diseases we find an acute broncho-pneumonia, not due to the entrance of the specific germs *via* the respiratory tract, but to the extreme degree of interference which the circulation suffers, and the consequent effusion of serum into the air tubes, especially the smaller, obstructing them and leading to

* These words were written before the receipt of Prof. Burrell's letter, May 1, 1889.

atalectasis and pneumonia. I neglected to call attention to this variety of consolidation in my report on swine plague, but it is really a very common form, and to be easily distinguished from that variety caused by the entrance of the germs *via* the respiratory tract, *which is more acute, more multiple, more rapidly caseous, and especially not hemorrhagic in any of its parts or in the surrounding tissue,* and also begins immediately around the tube, whereas this form begins in the more distant part of the brohchiolic territory. This is a point which I would especially call to the attention of some investigators in this country who fondly imagine themselves authorities upon questions of pathology, especially that of swine plague.

In the corn-stalk disease my experiences are unfortunately limited. I have found the form of broncho-pneumonia mentioned, and not the specific bronchial-infective type seen in swine plague. Considering that the manner of infection is of necessity by way of the intestinal canal, it is very doubtful if the bronchial-infective variety ever occurs in connection with this germ. But I have seen another form of broncho-pneumonia in this disease, due to an entirely adventitious bacillus capable of developing in the catarrhalic-infected bronchioles, but which had no more connection with the disease in question than if the animal had been afflicted with verminous bronchitis, and exposed to dust of some kind in which such a germ was suspended. These occurrences are more common in the septicæmic diseases of our domestic animals than the observers of this country and Europe seem to be aware of. In sections of such lungs, sometimes one variety and sometimes several varieties of adventitious germs, may be found mixed up with the genuine organism, which latter may have had nothing whatever to do with the lesions in question. Ultra-bacteriological investigators go on a wild-goose chase after such adventitious germs, while men of pathological common sense cast away such chaff, and pay their attention to the disease as it is, valuing complications as they should be appreciated. In this disease, as in swine plague, we may frequently find an entire absence of consolidation in the lungs, if I can judge by the very meagre notes sent me on the lesions observed, but still more by a correct appreciation of the natural pathological results in such a disease. In the same way with the spleen: sometimes it is swollen, often excessively, at others not. But never-failing lesions will be those of a haematogenic character, varying from capillary to coarse vascular

engorgements and hemorrhages of various dimensions. The diffuse capillary engorgement of the kidneys, which may be considered as pathognomonic of the southern cattle plague, (see plate VII,) never occurs in this disease, and hence may eventually prove of differentio-diagnostic value should it be finally shown that the germ of the corn-fodder disease also invades grasses and causes disease in animals grazing during the summer months. Acute lesions that will never be missed, aside from the disturbances of the circulation, are those of the dense parenchymatous glandular organs, which are essentially specific to the extra-organismal septicæmiæ, and vary in degree from clouded swelling to the most extreme grades of fatty degeneration, as has been shown in the very brief necroscopical notes.

To sum up then, the corn-fodder disease is an *acute extra-organismal septicæmia, due to a micro-organism belonging to the class of ovoid-belted germs, to which variety of diseases also belong the swine plague, southern cattle plague, Wild-seuche, hen cholera, and yellow fever in man, but in no case are these micro-etiological organisms one and the same, but each is a specific entity capable only of causing its specific disease, under natural conditions, in those animals specifically predisposed to its action, from some utterly unknown but equally specific physiological idiosyncracy peculiar to its species.*

SYMPTOMATOLOGY OF THE CORN-FODDER DISEASE.

In endeavoring to portray the symptoms of this disease, we come face to face with a question of exceeding difficulty, because of their very close resemblance to other diseases which occur in cattle in our Western States, and even anthrax itself offers intra-vital phenomena which very often more or less closely resemble those presented by this disease, especially in its most acute form. This fact has led many veterinarians into most serious errors in diagnosis.

In the firs tplace, as to duration. Like the swine plague, this disease may be fatal in twenty-four hours, or it may extend to eight or ten days before such a result occurs. It is not a universally fatal disease, and it is highly probable, is also of a non-recurrent type.

In the first place we have to do with an acute blood-poison disease, which, like all such diseases, is accompanied by a more or less excessive exacerbation of the bodily temperature, varying, so far as known, from 39° C. to 42° C., that is, from 102.2° to 107.6° F. Such an excessive

rise in the temperature must necessarily be followed by equally severe changes in the parenchymatous organs of the body, and consequent disturbances of the circulation, which frequently leads to excessive circulation-changes in the lungs, often followed by pneumonia, and most insufficient oxidation of the blood; hence, under such circumstances one would naturally expect a much accelerated and often very weak pulse and increased respiration, which, when consolidation of the lungs is present, is also very labored. (For a fuller account of these disturbances, see my report upon swine plague.) These disturbances of the circulation frequently extend to the brain, where engorgement and cerebral pressure occurs, which, in some animals, takes on the form of "craziness," as the owners call it. The animals then bellow fearfully, and chase other animals, especially dogs, hogs, or fowls, but seldom human beings. This has led to the mistake of their being called "mad" at times, and to this disease being mixed up with another entirely different one* by some veterinarians. Other animals stand by themselves, or are depressed and loth to move. Separation from their companions is one of the first indications of illness. As nearly as I can discern, they can all swallow and all drink. This is a very important point to be remembered. As in swine plague and southern cattle plague, constipation is a very frequent occurrence, while laxity of the bowels also often occurs, and may be looked upon as rather a favorable complication. Red urine does not occur. The visible mucosæ are injected, and often have a yellowish-red tinge. It will be remembered that my inoculated steer drank all the time, and even ate a little, during its most ill days. That such sick animals should be disinclined to eat, and often to drink, is no wonder; but if clear water is placed before them, no difficulty in swallowing will be discovered. Milch cows soon slacken in their yield of the lacteal fluid, and frequently the secretion ceases altogether for a time.

Diagnosis.—It has been already suggested that this disease cannot be mistaken for the southern cattle plague; first, because of the season of the year in which the latter occurs, and the absence of Texas cattle; and second, the longer period of approaching illness in the latter, and the general average of its duration, as well as the non-existence of hæmaturia (red urine) in the corn disease. Again, the outbreak in the corn disease extends more slowly over a herd, and continues

*See the next article.

longer under ordinary circumstances; or in other words, the certainty of exposure to infection of the majority of the animals in the same herd at one and the same time, is much greater in the southern cattle plague than in the corn-fodder disease.

That it bears no relation to the "Wild-seuche" of Germany is shown by the season in which it occurs, the locality and circumstances on which or under which it occurs, and the absence of enormous œdema.

In fact:

When cattle, (horses, or other herbivorous animals?) become unaccountably ill immediately after having been turned into a stripped corn-stalk field, and that illness is accompanied by the phenomena previously detailed, it may be taken for granted that it is this corn-fodder disease, and no other.

PREVENTION.

Prevention of the disease in corn and other fodder.—As Prof. Burrill's letter of May 1, 1889, has put us in possession of that practical knowledge by which we should be able to invariably tell when the growing corn in our fields is infested with this germ so dangerous to our live-stock, the question of prevention should be the simple matter of avoiding such corn-fields as places for turning in stock to do the post-harvest gleaning. From conversation with several farmers, I am quite convinced that even now some of them have quite distinct ideas of the manner in which the corn itself is affected, and that as we have obtained an exact description of this part of the story from the botanical side, we should now be enabled to totally prevent this disease in our live-stock, so far as the gleaning of our corn-fields is concerned. But this still leaves us to face several uncertainties which can only be settled by careful observation by farmers, and exact investigation by competent investigators.

First.—Knowing that the corn-fodder is diseased, the question is, Does the germ also penetrate the cob and growing kernel, and can they also cause the disease?

My own opinion would be to doubt it, had I not had a very singular experience with a single cob of half-grown corn. At the time I did not have suspicion enough to investigate, and was also very busy in other directions, and equally annoyed by the owner of a horse which ate some of this corn, so it was said, and it was assumed that the corn had been poisoned by a vicious neighbor. All that was brought me

was a "nubbin" ear of half-ripe corn. This was fed to a rabbit, and the animal died in three days afterward. There was every indication of a blood-poison disease. Anthrax was looked for, but not present. The organs were taken by the owner to a chemist, but nothing found therein. Cultures were not made, and only a very casual examination of the blood for bacillus anthracis, as I was busy beyond endurance at the time, and have never had any assistant to whom I could turn over the many such cases which have come to my notice.

This case is simply quoted as a warning worthy of the attention of other investigators.

Second.—Are grasses also infested by this germ? And if so, which? This last is a very important question; for if they cannot be, then such fields can be used for raising grasses. This can be best determined by actual experiment by intelligent farmers, who should turn such fields into grasses, including patches of millet and clover, and then feed a few cattle with each kind, without the admixture of any other herbaceous food.

It would be well if the experiment stations would make suitable arrangements in this direction with intelligent farmers, and bear the expense of using such an infested corn-field for experimentation in about the following manner:

1. It should be thoroughly cleansed of all refuse of the old corn crop, but not exposed to the action of fire.

2. A portion should be planted in corn, and if any stalks appeared diseased during the growing period, they should be fed experimentally, and under every precaution against accident from other causes, to cattle and also rabbits; the latter might be tried first, as enough is now known of the germ of this disease to enable any competent person to recognize it correctly. When the corn is ripe, the ears should be gathered carefully, and all full ears separated from those incomplete in development. Feeding experiments should be made with both. After the crop of corn had been gathered, a few cattle should be turned into it, and in order to avoid any error, exact methods taken to see that they were sufficiently supplied with water and salt.

Naturally each section of the field should be separated from the other, and if, as Prof. Burrill's letter suggested, such a field as one of "forty acres" can be pretty generally infested, such a one should be used for this kind of experimentation.

3. Various grasses, including millet, clover, and in fact all kinds used for feeding stock, should be planted in lots in such a field; one lot of each kind should be used for pasturage in the summer for a few cattle, while the crop of another lot should be preserved for winter use, and then fed to a certain number of cattle.

It is too much to ask any farmer to do all this at his own expense; but the interests at stake make it the imperative duty of the agricultural experiment stations to do it, this being one of the purposes for which they were created, though Nebraska is the single State that has done its duty thus far in this regard. There are an abundance of public spirited and intelligent farmers who will gladly support the work. In fact, Mr. McKelvie has written me that he intends to follow out my suggestions as to grasses and millet with the field he spoke of in his very detailed letter, stating his experiences with the disease.

Whenever such a disease has occurred, every stalk and leaf on such a field should be destroyed by fire, and until we know to the contrary, the field should be seeded down to hay. There is no question but what the infested remnants of the corn-fodder can, upon their decay, cause the further infection of the field by the germ thus becoming free and again developing in the soil.

Prevention of the disease in live stock.—In discussing this question I will limit myself to cattle, because they are the only species of our domestic animals in which the disease causes serious loss; in fact, the only one in which we know to an absolute certainty that it occurs.

Some might think it strange if I did not say a word about *treatment*, especially the farmer, while my scientific colleagues might think it equally strange should I do such a thing; for medicinal treatment, in any curative sense, is the height of absurdity in any disease of this class. Yet a close study of the clinical symptoms of this peculiar disease, and some knowledge of its pathology, does show that the offering of purgative doses of a saline character—Glauber's salt—to cattle in the very earliest stage of the disease, as well as to every member of a herd in which some have become ill upon exposure to this disease in a stalk field, is a matter of prophylactic importance. No harm can certainly be done by the thorough cleaning out of the intestinal tract in the animals still undiseased; and for those diseased especially, and perhaps also those in the early stages, such a method of treatment is most certainly indicated as a possible means of equalizing the dis-

turbances in the vascular system, and the avoidance of cerebral and pulmonary complications, as well as a tendency to deplete the blood of some of its septic elements, and check the supply by the removal of so many of the specific germs, as must necessarily occur in such a cleaning out of the digestive tract.

Such treatment, however, must take place within quarantined limits only, so that all the manure, and litter soiled thereby, can be destroyed by fire when the outbreak is over. It must be borne in mind that if the manure (and litter) from a cattle yard where animals have had this disease, is taken out and strewn over a field, and then plowed in, and that field is planted with corn, that such corn is very liable to become invaded by this germ, and can thus be the cause of more losses in cattle, if turned into such a "stalk field" the ensuing fall or winter.

On the eruption of this disease in a herd of cattle which have been used to do the lazy man's gleaning in a stalk field, the first step to be taken is the peremptory withdrawal of the herd from such a field and such fodder. The next thing to do is either to number, brand, or adopt some other means by which a record can be kept of each animal in the lot, and then take the temperature of each one night and morning. All with a temperature of over 100° F. must be looked upon as suspicious, and those in which it exceeds 102° F., as diseased. Those in which it does not exceed or rise over 100° F. need not cause any worry. There is no need of separating the sick from the well, as the disease is not contagious. As mentioned previously, a saline-purgative is indicated for all the animals. Those that die should be cremated, and with them a lot of the litter in the yard. If possible to avoid it, the regular cattle yard should never be used for such cattle after any of them have become ill. Again, I repeat, in no case should a particle of the manure or refuse from a place where such cattle have been confined ever be used for fertilizing purposes. *Burn it up, as well as the animals which die.*

ARTICLE III.

THE SO-CALLED "HYDROPHOBIA" IN CATTLE.

ARTICLE III.—*The so-called "Hydrophobia" in Cattle.*

THE SO-CALLED "HYDROPHOBIA" IN CATTLE.

What this disease really is I know not. The fact that I have probably discovered its germ—that is, cultivated one germ from several outbreaks, and produced apparently the same disease by inoculating healthy animals with pure cultures obtained from the cattle, (though not in cattle,) and have again derived pure cultures from each animal and found the same micro-organism in the tissues of these animals, as well as the cattle which acquired the disease in a natural manner—makes me most skeptical as to the disease being rabies, though in the outbreaks investigated by me in person, as well as numerous others reported in the daily journals during the past two and one-half years, the owners of the cattle have invariably reported the presence of a "mad-dog," and in some cases, especially the one visited at Dorchester, Nebraska, the owner and other persons most positively stated that some dogs that were bitten by the same dog which bit their cattle also "went mad." So positive were the owners of the cattle at Dorchester and Crete that their animals were actually bitten by a really "mad-dog" that it would have been the height of folly for any one to have persistently argued against it; and it must be freely admitted that I have neither argument or evidence of any value whatever against such positive testimony on the part of these owners. On the other hand, when I reflect that for a number of years unquestionably competent investigators in various parts of Europe, many of whom have never been heard from because of their negative results, have been diligently searching for a specific micro-organism in the tissues of animals which were called "rabid," but especially in those of dogs, as well as in the materials and virus prepared after Pasteur's method, and knowing the peculiar nature of the disease, I can do nothing else than doubt that this disease is rabies which it has been my fortune to study. On the other hand, it should be mentioned that I have been unable to find a "mad-dog" since I have been in Nebraska, though the number of

"mad cattle" reported has by no means been small. In the light of the total absence of any attempt to gather reliable statistics as to the prevalence of devastating diseases among the live-stock of the State or country, and placing my judgment entirely upon newspaper reports, it looks as if this disease causes fully as much loss to the cattle owners of the State as all others combined, with the exception of the previously considered "corn-stalk disease." In fact, it can be seen by turning to the quotation from the report of the late live-stock commission that the worthies composing that body looked upon this "Hydrophobia" in cattle as identical with the "corn-stalk" trouble; without for a moment noticing that the latter, according to their observations, occurred only in the late fall and early winter months, while the reports of this "hydrophobia" complaint, though occurring in the summer or hot months, have also been made in all seasons of the year. As has been said, there are no statistics of any kind accessible upon this subject, except such reports as are frequently telegraphed to the daily papers. A selection of a very few of these will at once show that the disease spoken of cannot possibly form a unicum. One has already been isolated from this complicated mass in the preceding article.

As examples, I quote the following cases:

1. "DeWitt, Neb. For several days dogs, sheep, cattle, and horses have been afflicted with hydrophobia, and the fatality has been great. Within the last few hours the ravages have increased to an alarming extent. One hundred and eight sheep have died with the malady, and the disease is rapidly spreading." December 1, 1885.

That the above report had no connection with rabies is self-evident. It is highly probable that the report that dogs were dying also was a creation of the imagination. The story looks more like the corn-stalk disease than anything else.

2. *Hydrophobia in Horses.*—Six head of horses were afflicted with what the veterinarians called "hydrophobia." The reporter says that "they could not look upon water without becoming frantic, and refused all food; each animal had bitten its own legs and sides as if suffering the most terrible pain." February 2, 1886.

3. *Hydrophobia in Sheep.*—A. S. Bebout, Coryell county, Tex., writes: "I cannot tell you what three dogs can do with a flock of sheep in a day and night, or any other time, but I can tell you what one

dog did for one of my neighbors. He had 1,550 sheep in a corral containing about one-fourth of an acre. A dog entered his lot on the night of October 28th. No one knows how long it was in there. It did not kill any sheep outright. Seven or eight were badly wounded, and many others were bitten, but their wounds were too slight to be noticed. Twelve days afterward they began dying with hydrophobia. They would live all the way from five hours to ten days after showing signs of disease. The first signs were frothing at the mouth, butting, or biting, at the other sheep, or pulling their wool. Some of them would catch hold of a man's coat-tail and hang on like a dog. Ninety-five head died within two weeks after they commenced dying. The last one died March 28th, and up to that time 112 head had died. All died of hydrophobia, and on all could be found a mark to show where the fatal tooth had struck."—*National Stockman, May, 1889.*

And the following from a local paper:

"Hydrophobia carried off a large number of hogs and cattle belonging to a Hamilton county farmer."

"A fine short-horn cow, owned by H. Walton, near Unadilla, while suffering from hydrophobia, attacked a splendid thoroughbred Clydesdale mare belonging to Mr. Walton, and in an instant the animal was torn from flank to foreleg, causing instant death. The cow died a few moments afterward. E. Luff, a neighbor to Walton, lost a splendid Clydesdale mare from hydrophobia."

Quite a number of cases where the animals have been so mad as to tear their own bodies have been reported to me, but I have not seen any in person, with the exception of one, in which the animal was frantic, and had torn all accessible parts of its body in a terrible manner. The only thing of any value about this case was possibly the fact that it was pastured in a lot through which ran a half-dried-up stream. I was too busy to make any examinations, the case only coming to my knowledge by accident.

This disease may have been the somewhat notorious "mad itch," which has been attributed to most anything and everything, but which Dr. Paquin, of Missouri, has shown to be transmissible by inoculation, and not due to any of the various hypothetical causes popular among the people. It is to be regretted that Paquin gives us a detailed description of neither the clinical nor microscopical phenomena, for both of which he seems to have had ample opportunity and material.

It is also singular that he makes no mention of any attempt to discover the germ.*

One thing is very sure, and that is, that aside from the "corn-stalk disease," in which cerebral excitement is frequently present, there are other diseases in live-stock in the West in which "mad" phenomena are very common, and in which a "dog" is not always the sensational accompaniment.

It may be well to call the attention of the reader to the fact that some changes will be found in the following remarks from the text of the communication published in 1886.

Probably one of the best studied outbreaks of this reputed "hydrophobia" in cattle was the one observed at Crete, Neb., in the summer of 1886, simply because of the rare opportunities which the case presented for protracted consideration.

HISTORY OF THE OUTBREAK AT CRETE.

About 4 P. M. on Saturday, July 17, 1886, a stray dog, said to have been a shepherd, was observed crossing the highway, about seven miles from the town of Crete, and to enter the adjoining pasture, in which were a large number of grazing cattle, mostly two and three-year-old steers. The dog is reported to have at once dashed in among the cattle, chasing and worrying them with great pertinacity, though the cattle were in no ways backward in chasing the dog, but did not succeed in driving him off. He was sseen to bite several of them, and indeed to catch some of them by the nose and throw them down. The farmer who observed the dog enter the pasture, and its very singular and vicious actions, at once sent word to his neighbors, some of whom owned the cattle, so that in a short time quite a number of persons had collected. The steers in the first mentioned pasture frequently succeeded in driving the dog into the next pasture, where there were cattle also, which the dog bit and worried in the same manner. In this way the sport went on for about two hours, notwithstanding the efforts of the men, some of whom were armed with revolvers. The dog seemed to find so much more to attract its attention in the cattle, that it did not offer to attack the men present, only taking notice enough of them to try to evade them.

I have since learned that the dog also bit two dogs on its way to the

*Bulletin No. 33, Missouri Agricultural College.

pastures, and one calf, which will be noticed presently. The two dogs were immediately killed. On November 5 I was also informed that still another dog had been bitten by this dog, which was not killed until recently, when the owner observing it to be "mad," shot it. This must be taken *cum grano salis*, so far as the "mad" is concerned.

It is of the utmost importance, in the consideration of this outbreak, to know that these pastures bordered upon the Blue river, and that all along its banks were numerous pastures, in continued succession in both directions from the two in which the cattle bitten and hazed by the dog were grazing. Though the grasses, weeds, trees and shrubs, as well as the river water, were all the same in all these pastures, still not a single animal of the bovine species died of any other disease in any of the other pastures, or among the cattle which were removed from them during the balance of the grazing season of that year, except one cow that died from an undoubted attack of puerperal eclamsia, and one calf that was in a field which the dog must have passed over in order to get into the pastures mentioned.

Mr. Imhoff, who owned the calf just mentioned, is a very intelligent farmer, and reported the following:

The calf was six months old, and perfectly well up to the time of the accident. It was constantly kept in a small pasture near his house. He observed the dog come into this pasture and make at once for the calf, which it worried for a few moments, and then left it, going out of the pasture in a direction leading directly to the previously-mentioned pastures. The calf was taken about August 15th, and lived nine days.

Symptoms as reported by owners of the cattle.—Mr. Imhoff reports that his calf showed great irritability and uneasiness; wild-appearing eyes; constipation, with repeated straining in the earlier days of the illness, terminating with a tendency to diarrhœa, the fæces, however, being only passed with difficulty, and in small quantities; the urine was passed frequently and also in small quantities; desired to eat and drink, but could not swallow; masticated food, dropping it again out of the mouth, and the fluid flowing out of the nose; was not unduly excited at the approach of those with whom it was acquainted, nor by the presence of the house-dog, to which it was accustomed; but on a flock of ducks getting into the field, rushed upon them and stamped

and tore several to pieces before they could escape. It finally became paralyzed in the posterior extremeties, and greatly emaciated.

The other owners report similar phenomena in their cattle. They were very much excited and uneasy, and chased the other cattle around with great fury, horning them frequently, and bellowing furiously nearly all the time. All noticed the peculiar hoarse, unnatural tone of the voice. They were all constipated, and strained much in their endeavors to pass manure; some became diarrhœic, others not; urine was passed frequently and in small quantities; they would try to eat and drink, but would soon give it up, for the time, in apparent disgust. They could not swallow, and the half-masticated food or drink would either be dropped from the mouth or flow out of the nose. All observers report that the diseased cattle were not exceedingly excited by the presence of the men who salted them, but that if strangers came into the field, they would chase them furiously, and in several cases such persons had to climb trees to escape them. When confined they would stand listlessly in one place for a long time; but occasionally, or when the other cattle approached the pen, or human beings came near them and made any unusual movements, they would rush furiously and blindly against the barriers. The eyes were wild, and the pupils wide open. They all became more or less emaciated, and those which were not killed, paralytic, sometimes in the fore, but more frequently in posterior extremities. The cows which were affected rapidly ceased to secrete milk.

Mr. Gilbert White, of Crete, one of the sufferers, kindly sent me the following list of the animals which died or were killed:

One became rabid August 4, twenty-one days after the bite.
Four became rabid August 8, twenty-five days after the bite.
Five became rabid August 10, twenty-seven days after the bite.
Three became rabid August 15, thirty-two days after the bite.
One became rabid August 19, killed by me, thirty-six days after the bite.
One became rabid August 24, forty-one days after the bite.
One became rabid August 29, killed by me, forty-eight days after the bite.
One became rabid September 14, fifty-eight days after the bite.
One became rabid October 5, seventy-eight days after the bite.
One became rabid October 10, eighty-three days after the bite.
One became rabid November 3, killed by me November 8, 108 days after the bite.

Suggestions excited by the above statistics.—Before entering upon consideration of my personal observation of the clinical and necroscopical phenomena in these cases, I wish to call attention to one fact which made me very skeptical as to the rabies theory in connection with this very striking outbreak. Nevertheless, the most critical observation of everything connected with the animals, and a most exact cross-questioning of the owners, could not bring out a single moment which would speak for any other hypothesis.

This fact was, that so many animals should have died at so nearly the same time, with such short intervals between the deaths, there having been in one case three deaths on the same day, and that thirteen cattle and one calf should have either died or been killed within a period of thirty-eight days from the day upon which they were seen to have been worried by the dog. From some very casual studies of the literature at my command, I have made a list of 400 cases of reputed rabies in cattle. The average period of incubation in these cases was forty-four days, the shortest being nineteen.

I could scarcely make it conform to our knowledge of this disease that so many animals should perish in such a short period, and nearly at the same time, after being ostensibly bitten by one and the same dog. I therefore reported to the owners, at the time of my first and second visits, August, 1886, "that there was no other moment upon which we could place any etiological support, and that I considered they were justified in adhering to their mad-dog theory, and that I must admit that if the cases were not rabies, that I did not know of a single bovine disease which, considering the history, the phenomena were conformable to."

In favor of the rabies hypothesis we have the following conditions:

1. The cattle in the same lot which died since then all presented phenomena which correspond in all essentials to those seen in the others, as well as in every particular to those given in text-books for bovine-rabies, except that none of these animals were seen to bite or gnaw any particular spot of their bodies.

2. The intervals between the deaths having become more extended, is still more evidence in favor of the rabies hypothesis.

3. As other cattle had been placed in the same pastures with the diseased ones and other cattle that were there at the same time, both during the outbreak and since, there could have been no common local

cause of an infectious or intoxicating nature in the pastures, as there have been no deaths among the cattle since from other causes.

4. The case seen November 5 presented the same symptoms that the others did; but what is of vast importance *is, that 108 days had elapsed since the dog had attacked the cattle, and this one was among them at that time.*

Personal observations.—I will now give the observations presented by this last animal, which was first seen November 5, 1886, as they bear directly on the above statements. Mr. Vance, the owner, reported that the first thing which attracted his attention to this animal the third of November, was "its very nervous and irritable condition. It was much excited, and bellowed furiously with the peculiar changed voice noticed in his other cattle; it would rush at and horn the cattle and tumble the hogs round right and left, and then stand panting and exhausted for a time, when the same maneuvers would be commenced again. Its eyes were wild and distended, and it frothed * at the mouth. It could neither eat nor drink, though it would take food and water into its mouth as the others had. It was constipated."

The animal was lassoed with some difficulty, and placed in the scale pen, where we found it on the morning of the 5th. It at once became more quiet, unless approached by strangers or the pigs, when it would charge furiously against the walls of the pen.

Status-præsens.—Red steer, two years old, somewhat thin, standing in a pen over the scales. It remained quiet until I suddenly rushed by its pen, when it made a furious rush against the barriers. The pupils were distended in the perpendicular diameter; the eyes were wide open, and had a peculiar wild look in comparison to those of other cattle standing in a neighboring field. It had only passed manure once since yesterday, but urinated several times in the twenty minutes we had it under observation. The manure was dry and hard, and passed with difficulty in a small quantity.

The diseased cattle possessed abnormal tastes, and would frequently take the manure into their mouths and chew it, and try to drink their own urine. Mr. Vance reported the same in this case. Pulse and temperature could not be taken; at least it was not prudent to try it on. On driving some pigs up near the pen, the animal charged upon

*This "frothed-at-the-mouth" statement must be taken *cum grano salis.* The animal simply could not swallow, and when excited champed its teeth, which naturally caused some froth in the accumulated salivary secretion.

them furiously; when driven away again, it soon became quiet. On corn being given it, it would take hold of it greedily, but soon stopped masticating as if from disgust, and allowed it to fall from its mouth again.

On a pail of water being placed within its pen, we observed a most singular phenomenon — one which has never been described before, so far as I know, and which was not noticed in any other of the affected cattle at Crete.

The animal dipped its nose deeply into the water, and tried for a moment to drink, but finding it impossible to swallow, with a really intellectual perception of its own condition, it became more furious than we had seen it, or I had seen any of the others in confinement, and attacked the bucket until it had smashed it to bits. It was only quieted when we removed the remnants through the fence with a fork. The owner reported the same actions on the part of the animal every time he offered it water.

If the above was not intellectual action — the desire to remove an offending yet much desired and irritating object out of sight — I do not know what it was. It exactly corresponds to the phenomena seen in human beings in reputed cases of rabies, and shows still another thing, viz, that *there is no such thing as hydrophobia!*

Man does not fear water any more than this steer did. He does fear the pain of swallowing, or, what is worse, he fears that in attempting to swallow he will choke to death.

This steer could not have any conception of the latter, but it had gained an intellectual perception of the fact that it could not swallow: there was no evidence of pain, however, and knowing that, it smashed the offending bucket to pieces, just as a man, under the same circumstances, would dash a glass of water against the wall or on the floor, with violence enough to smash it to pieces. The poor animal had not been able to swallow a drop of water for over thirty-six hours.

The other phenomena observed in this animal correspond in every particular to those seen in the other cattle, and certainly point to a common cause for the disease in all of them.

The animals seen previously were two in number, and were killed at my request August 19 and 25 respectively. They belonged also to Mr. Vance's herd, out of which nine cattle had already died, the one alluded to above being the tenth which he lost.

The first animal was in a very strong pen, built especially for the purpose, at one end of the pasture in which the balance of the herd were grazing, but out of sight, on account of a grove of trees. This animal was a red steer, two years old, and was in a fair condition, but was said to be emaciating very fast. As the pen was approached from a distance, we stopped to observe it. It at first stood still, but bellowed almost continuously, the voice being hoarse and of a peculiar rasping dullness. This bellowing increased as it heard our approach. As we neared the pen and made any unusual movements, it would rush furiously toward the point from which the noise came until it brought up against the timbers of the fence. If we remained still, it would cease to move for a few moments, and then move listlessly and apparently blindly from place to place. Its movements at such times were weak and tottering, with crossing of the posterior extremities and dropping of these parts. At other times it would knuckle forward, or even fall on its knees, but would immediately recover. One eye was amaurotic. The pupil of the other could not be seen, on account of cloudiness of the corner from injury from the wire fencing between the timbers of the pen. When caught it was reported to have been extremely wild and ferocious, especially to several strangers who were in the field, who had to climb trees to escape its charges. So far as is known, it was the fifth day of its illness when I saw it. It frequently attempted to pass manure, but only succeeded in evacuating small masses, of a pultaceous consistency. The passages of urine were frequent and in small quantities. On food and water being placed before it, it would try to eat or drink, but soon gave it up, the food or water escaping again, by both the nose and mouth.

Necroscopical observations.—No outside markings worthy of note. On cutting through the skin, a purple-blue-red fluid exuded from the cut vessels, which soon coagulated and became red on contact with the air. No exudation in the abdominal cavity. Bladder empty. Peritoneum abdominalis normal. The small intestines were of a pink-red color; the larger ones somewhat so. Mesenterial blood vessels engorged with blood, as well as those of the omentum; the mesenterial lymph glands were swollen, the parenchyma, on cross-section, being juicy and of a pink-red color. The spleen was somewhat enlarged, but not degenerated, nor did it contain an abnormal quantity of blood. As the disease had been pronounced to be anthrax by some persons, I

had provided myself with a suitable microscope to test that question. Bacillus anthracis not in the blood.

The liver and kidneys were somewhat swollen with blood, but there was neither clouded swelling nor parenchymatous complications, so far as a microscopical examination could determine.

The fauces were swollen and covered with a viscid substance; large vessels injected; larnyx swollen and vessels engorged. The clinical phenomena indicated that there was paralysis of the muscles of these parts as well as of the œsophagus.

The œsophagus presented a singular appearance, being distended from its superior ostium to about the size of a Bologna sausage down to a point corresponding to the curve of the posterior aorta. On opening it, it was found to be completely filled with partially masticated corn and fodder, which terminated in a bolus of the latter at the point mentioned. This bolus was not large, and could have easily passed on to the rumen had it not been for the lamed condition of the muscularis. The same clinical phenomena have been seen in all other animals with regard to swallowing either food or drink, but this condition of the œsophagus was not seen in the other necroscopies. Still I do not think it had anything particular to do with the ability of this animal to swallow more than others, but rather to have been due to an extreme degree of paralysis of the muscularis of this portion of the digestive tract very early in the disease, which caught the food, as it were, in its passage toward the rumen.

Lungs, heart, and pleura normal.

The rumen and reticulum were well filled with ingesta, in a somewhat dry condition. The omasum was the hardest I ever saw, both to the touch and in its resistance to the knife; the interlabial spaces were completely packed with very dry material. No signs of inflammatory irritation were to be seen in the linings of either of these anterior stomachs.

"DRY MURRAIN."

Abomasum empty. The mucosa was somewhat swollen, and of an intense deep pink-red color, but free from catarrhal effusion or hemorrhages. The mucosa of the small intestine was likewise of a diffuse pink-red color, but not so intense as that of the fourth stomach, while that of the small intestine was still less so.

The contents of the large intestine were semi-fluid, but became

thicker and thicker as one approached the rectum. On opening the brain (which should always be done first, but in this case the description comes last) there was found to be a considerable quantity of reddish aqueous fluid between the pachia and lepto meninx. The vessels of the latter were distended with blood; the large sinuses were in the same condition. The gray substance was of a moderately deep pinkish red, and even the white had a somewhat reddish tinge. The cut surface was moist and glistening, and disturbed by numerous ecchymotic spots which extended diffusely into the surrounding tissues. A dark-colored fluid oozed from the cut vessels. Each of the lateral ventricles contained over a tablespoonful of a red-aqueous fluid; the vessels of the Choroid plexus were much distended, and of a dark red color.

The medulla oblongata was marked by an unusual degree of redness, and a lustrous appearance of the cut surface, which was disturbed by numerous diffusely terminating ecchymotic spots.

The results of the autopsy made August 28 were exactly the same with the exception of the absence of the stuffed condition of the anterior portion of the œsophagus previously mentioned.

Necroscopy on the steer seen November 5, 1886.—November 8. Took the train early this morning, accompanied by Drs. Bowhill and Thomas, anticipating that Mr. Vance's steer would be much worse. Found my anticipations correct; as Mr. Vance had just harnessed his horse to go to town and telegraph me. He reported that the steer had become much worse, and very furious at times, having injured one of his arms with its horns when trying to place a bucket of water in its pen.

Status-præsens.—Marked signs of emaciation since we last saw it on the fifth; had not eaten or drank in the mean time. Eyes bloodshot and wild looking; veins of retina markedly injected. Very excited upon the least movement on our part, and bellowed every other moment, stamped with fore feet, and tore furiously around the pen. On a small water-trough being put in the pen, it attacked it with intense fury, so much so that we put poles in and distracted its attention so that we could remove it, as there was great fear that it would break the barriers down. Urination frequent.

Shot through the heart by Dr. Thomas.

While standing and bleeding it passed about a pint of thick, pul-

tacous manure, to which it immediately turned, and tried to eat it; soon dropped in its tracks.

Necroscopy, by Drs. Bowhill and Thomas.—Brain.—On removing the cranium the large sinuses were found filled with a blue-red, semi-coagulated fluid. The vessels of the pia mater were very much distended with a dark blue-red fluid, and extended above the surface of the membrane. The gray substance had an abnormally red shade; cut surface moist and glistening; the same for the white substance. At the base of the brain was a considerable quantity of straw-colored fluid. The ventricles contained a quantity of a red, aqueous fluid; vessels of the plexus distended. The medulla oblongata was surrounded by an abnormal quantity of a fluid of a straw color. The longitudinal veins of the spinal canal were found distended by a dark blue-red fluid. The cut surface of the medulla was moist and glistening.

Oral Cavity.—The fauces were some swollen, and covered with a viscid material, the large vessels being injected. The entrance to the larynx was reddened and very much swollen, but the mucosa of the trachea was only swollen a very little, though the vessels were considerably injected; same of the bronchial tubes.

Bronchial lymph-glands swollen and somewhat reddened; cut surface moist and glistening.

Lungs.—Normal.

Myocardium.—Normal.

Abdominal Cavity.—Blood dark blue-red. Vessels of omentum and mesentery enlarged. Some diffuse redness in spots in both membranes.

The outside of the small intestine was of a delicate diffuse pink color; that of large less so.

Spleen.—Was the shape of an English "sole," oblong, oval, both ends having about the same diameter, fifteen inches long, five inches wide at one end, four and and one-half at the other, one inch thick on an average.

Liver.—Swollen; opaque; clayish-gray in color; gall bladder moderately full.

Kidneys.—Somewhat swollen; cortical substance of a yellowish-gray-red color; opaque, anæmic; medulla reddened; vasa recti very plain.

Bladder.—About one-third full; urine of a pale straw color; albumen present in small quantity, (tested at laboratory).

Stomach.—Only partially full of ingesta; linings of first three somewhat softened, and pealed off easily. The third stomach was but partially filled; contracted. There were many small stones in it. Mr. Vance remarked that "All his sick cattle had shown much fondness for earth." Fourth stomach—Mucosa swollen, and of a pink-red color, interspread by numerous ecymoses of variable dimensions; here and there were darker and more extensive hæmorrhagic centers. Intestines somewhat contracted; contents of small intestine semi-fluid, but not very much of it; that of the large became thicker and thicker as the rectum was approached; the mucosa was not much swollen, but somewhat reddened; large vessels injected.

Mesenterial lymph-glands swollen, moist, and diffusely red.

AN OUTBREAK OF THE SAME DISEASE AT DORCHESTER, NEB.

On August 21, 1888, a rumor came to me that a Mr. Andrew Moffat, of Dorchester, was losing cattle from the so-called "mad" disease, and I immediately wrote him for more positive information. On the 25th he answered that he "had already lost five cows and three hogs, having killed them as soon as they became wild, for fear they might do some damage." He had none ill at the time, but on August 31st he wrote again that "another cow was sick, and that he would try and keep it until I could get there," which occurred September 4th.

It may be well to remark that the town of Dorchester is near Crete, and that Mr. Moffat lives but a few miles distant from the aforementioned Mr. Vance. His farm, however, is not near the Blue river, nor has it any running streams, being rolling but elevated and dry prairie land. The stock received their water from wells. Attention should also be called to the fact that no disease of this character had occurred at Crete or in the vicinity since the outbreak at about the same season of the year in 1886.

A dog in the story.—According to Mr. Moffat, the dog which bit the animals began to act strangely on the 27th of June, becoming very much excited, and roaming over the country. It was killed June 30th. It is reported to have bitten quite a number of dogs in its ramblings, some of which are reported to have "gone mad" and been shot since.

These statements were given me by Mr. M. as facts, and supported by similar assertions from quite a number of neighbors present. As there were still said to be a number of dogs in the vicinity that had also been bitten by the "mad" canine, I did my best to have one such, or more, sent by express to the station, but, as in every other such case, the desired dog has never come. It may be asked why I did not attend to it myself. To which I answer that I had all I could do at the time to make an autopsy and cultures single-handed, and get back the same day. In cases like this, the public, while desiring to help, are next to useless for fear of getting infected, and one has to do all the work alone, where there is any danger of touching the animal or anything which had been in contact with it. It is singular what the effects of ignorance and superstition are among our farmers. There are but few among them who would not at once skin and cut up an animal that had died of anthrax, though they knew nothing about it, which is a thousandfold more dangerous than to perform the same operation on these "mad cattle," where it is hard to get them even to hold onto a rope tied to the animal's feet to keep the body on its back, though the end in their hand has never been near such an animal. Just here let me say to the farmers, that it is well they find out what anthrax is, for it is about the only disease of their stock which it is absolutely dangerous for them to have much to do with. Glanders is by no means as dangerous. Like rattle-snakes, fortunately anthrax is rather rare.

To return to the dog.

The "mad dog" had been trained to drive in the milch cows. It was certainly observed to bite one of the cows which subsequently became ill and was killed, according to Mr. Moffat, his sons, and hired men. Seven head of cattle were killed in all, but this was the only one positively seen to have been bitten by the dog in question. On the other hand, it was asserted that the dog was seen to bite two pigs and an old sow, all of which died presenting phenomena indicatory of cerebral excitement and "fits;" they were also said to have been dangerous. As to the cattle, they were all reported to have acted in about the same manner, and as Mr. Moffat expressed it, "Just as them Crete cattle did when you were over there, doctor." Some were more furious than others; all were dangerous if excited, and horned the other cattle badly; but nothing seemed to make them quite as wild as

to have the hens, or fowls, or a hog, but especially a dog, (natural in all cases,) get in where they were. Their eyes were reported as "looking wild and bloodshot." They could not swallow, but would try to both eat and drink, on either food or water being offered them. They would chew the food, hold it a little, and then let it fall from the mouth. When water was given them they would "ram their noses into it, and then get mad and smash the bucket, unless they knocked it out of reach." They were all severely constipated. Each animal kept up a most continuous and peculiarly-unnatural bellowing. From the first it did not have the healthy ring of the true bellow of cattle. They emaciated fast, and became "weak in the legs, especially the hind ones." The majority of these cattle were milch cows, and a singular phenomenon, (what self-evidently could not be seen in the Crete steers, but which may have, or may not, differential-diagnostic value to future observers,) was the fact that though all the cows were "in calf," and some soon to "come in," each and every one of them became "bulling" in a most frantic manner. This condition has never been reported in either the corn-stalk disease, in which the cattle are "mad" enough sometimes, or the so-called "mad itch," but has been in other cases where a "mad dog" was in the story. I wish it to be understood that I am not writing *for rabies*—rather against it; but what I am trying to do is to show that we have here an idiopathic malady marked by some quite distinct phenomena.

PERSONAL OBSERVATIONS ON A COW KEPT FOR ME AND KILLED AT MY REQUEST AT DORCHESTER.

The animal, an aged but well-bred cow, was very wild, and so dangerous that Mr. Moffat scarcely felt safe in keeping her until my arrival. It was confined in a shed, and tied by a heavy chain to a very strong post directly against but outside the wall of the building, the double chain passing through the same. Eyes wild and conjunctiva injected. Had been ill some days, and was much emaciated. Showed excessive signs of œstrum, though in calf; movements nervous, excited, and uncertain; thrashed round considerably. A peculiar phenomenon was a sort of string-halt-like twitching of the posterior legs and automatic vicious kicking; but not directed at anything. Between the intervals one could pass safely behind the animal, and even when standing near and its attention attracted, it was as lia-

ble to kick with the other leg as the one nearest such a person. Was and had been excessively constipated. Passed urine not only frequently, but involuntarily, and in small quantities. Bellowed almost constantly. When tried with food and water it dipped its nose with the greatest lust into the bucket, (in fact, whether it was imagination on my part or not, it did seem as if I could see an expression of most extreme desire for water in the poor brute's eyes as the bucket was set before it,) but was unable to swallow a particle, though it filled its mouth, the water again flowing out by the nasal openings. Finding itself unable to swallow, it repeated the operation mentioned in the case of Mr. Vance's steer at Crete—it smashed the bucket to pieces and went into a frenzy over it. It then tried to drink the water on the ground, and on being unable to swallow that, it began to horn the ground as if to vent its spite in that direction. In fact, the actual sufferings of the animal on account of its inability to swallow cannot be portrayed in ordinary language. On food being given to it, it masticated the corn with avidity for a few moments, then tried to swallow, and on finding itself unable, stood as if either disgusted with its efforts, or paralyzed with astonishment at what it could not understand. Except when excited for some time, its respirations were but little accelerated, and never labored. Neither pulse nor temperature could be obtained, except at too much risk of a broken limb.

After examining it all that the time at command would allow, two long ropes were tied to its horns, one as a check being taken backward to a tree, and the other to another in the direction in which we wished the beast to go. The chains were then slipped from its horns and it rushed blindly in any direction. On being checked, and the ropes slackened, it did not seem to see any one standing within a few feet, but would rush off toward a noise made by any one else in an entirely different direction. Its eyes were amaurotic, which could not be distinctly seen before in the rather dark shed. After testing it several times in these ways with the same result, the animal was securely tied to a tree, and shot through the heart.

The time was growing short, and the chief purpose being to obtain cultures, I only took a hasty view of the organs as I removed them, my difficulties being unnecessarily multiplied from the fear of those around me to touch a hand to the animal, or to any tool which I had touched.

Necroscopical Notes on Dorchester Cow.—Blood of a purple-red color, and about normal consistency, oxidized soon upon exposure to the atmosphere; that within the thoracic cavity, on account of the way the animal was killed, was completely solidified. Fat of omentum of a peculiar reddish-yellow, (I do not know as this point is of any value whatever; but while not constantly present, I have observed it in these cattle, as well as in a horse, which was said to have been bitten by a "mad-dog," and did die under almost the same symptoms as the cattle, the fat from the omentum being all that was brought me, and that only on account of its peculiar-orange-red color,) as was also the kidney fat. Mesenteric vessels much engorged, and lymph-glands swollen and very juicy; serosa of the small intestine slightly swollen and clouded, and of a dull pinkish-gray color, interspersed by an occasional petechial or more extensive and somewhat diffuse hæmorrhagic center. Nothing necessary to comment on in the first two stomachs, except that they contained an abundance of food and a good supply of water. To the "Corn smut, corn stalk, short-of-water-and-salt, dry murrain" fanatics I would say that here was their paradigmatic "dry murrain," the third stomach being hard as a cannon-ball and contracted and full of dry food, and all their conditions fulfilled except the "corn smut and corn stalk" portion; but there was water enough present to have lasted the cow several days more, though she had not drank for three days. Her food had been grass, and she had been salted regularly; and yet, strange to say, she died of "dry murrain," or would have, had one of these wiseacres made the autopsy. The fourth stomach could naturally contain but little food with the third in a condition forming a sort of dam before it; its mucosa was, however, very much swollen, and of a carnation pink-red color, its diffuseness being relieved by quite a number of hæmorrhagic centers which varied in extent and form. Mucosa of the small intestine also swollen, especially in the duodenum and anterior half of jejunum, then decreased; here it was also pinkish-red, and like that of the stomach, covered with a viscid secretion. *Spleen,* normal in size and consistency. *Liver,* somewhat swollen; parenchyma of grayish-red color, clouded, slightly anæmic; peripheries of acini slightly yellowish-gray, centers red; gall bladder full. *Kidneys*; swollen, cortex pearly-gray color, opaque and anæmic; medullary injected. *Lungs,* normal; bronchial lymph-glands, swol-

len and juicy. Mucosa of pharynx much swollen, and thickly covered with a viscid material; glottis in same condition, which extended through the œsophagus. A quantity of semi-masticated corn in pharyngeal cavity, but not a grain along the canal of the œsophagus. *Brain.*—Vessels of pachia-meninx excessively engorged, with hemorrhagic centers scattered through the membrane, as well as in the lepto-meninx and through the substance of the brain, which was unduly pinkish in color, and œdematous. Apices of the anterior lobes were the seat of quite a considerable extravasation. Plexus of ventricles injected, but no effusion present.

Tubes were inoculated from the plexus, brain, liver, spleen, and blood, and material was taken out with sterilized knives and placed in sterilized bottles for examination while fresh. At the same time other material was at once placed in alcohol.

THE GERM OF THIS DISEASE.

Whatever the definite diagnosis of the real nature of this strange disease may be, I am quite certain that I have discovered its germ. From the animals killed and examined August 19th and 25th, 1886, respectively, I did not obtain the satisfactory development in the tubes which I did from the one killed November 8th of the same year, and especially from the Dorchester cow in 1888. The first tubes were inoculated in the open field from the vascular rété in the cerebral ventricles, brain substance, and the medulla oblongatas. As was to be expected under such circumstances, quite a magnificent display of fungus-flora resulted in a large number of the tubes, but even among this material there developed and grew out colonies of a germ which presented itself in pure cultures in quite a number of the remaining tubes, and which gave ample material for examination, as well as some very striking results in some inoculated puppies.

Results following inoculations of puppies.—Before describing the germ I will briefly relate the results following subcutaneous inoculation of pure bouillon cultures in puppies. At this time, and even while studying the outbreak at Dorchester, I did not have a proper place to keep dogs in safety which one has to do with a disease in which a strong suspicion of rabies exists; so that I had to take small puppies that could be confined in wire cages. Let me say that in none of these experiments did I have recourse to the Pasteur or intra-

cranial inoculation, for the simple reason that it does not seem to me to be a natural procedure. Whatever the disease may be, I am of the opinion that such test experiments must be as near an imitation as possible of the manner in which infection occurs under natural conditions; and without discussing that point in general any further, I will say that in rabies the only just and proper way to demonstrate either that a given germ derived from suspicious material, or a virus of any form whatever, is specific, is to follow the way of accidental infection as it occurs in nature—that is, either by the scarification of the cutis, or by cutting through it and then introducing the material, or the subcutaneous injection of the same.

In order to show that one has the specific germ, or even a specific virus of rabies, the disease must be caused in a healthy dog in one of the above ways, and then it must be demonstrated that it was genuine rabies by exposing healthy dogs to the bite of such an inoculated dog; and natural rabies must result in such bitten dogs, which latter must extend the disease to other dogs, by biting them, and so on, following nature's course.

In exogenous or blood diseases, where the hæmic fluid is replete in germs, and where, in almost every case which is examined before any cadaveric changes can possibly occur, as can be done in anthrax, swine plague, the southern cattle plague, (Texas,) and yellow fever, we can or should find them in a pure condition, and a diagnosis as to the germ can be safely made. In endogenous diseases, however, especially one like rabies, we must have experimental proof of the most exact kind, or else such undoubtedly clear proof as to the presence of the germ that there can be no question of its specific relation to the disease. In this case I feel convinced that I have established that point as to the cattle, but have no evidence, other than the historical, that the micro-organism discovered has any relation to genuine rabies. Having come back to rabies again, let me also say that to my mind the first question one must ask and find demonstrated by continued and unquestionable evidence before he is warranted in taking any steps toward prevention by artificial inoculation, is: Under natural conditions is the disease in question generally non-recurrent; and also, is a mild attack as sure in producing this non-recurrent condition as one in which a patient barely escapes with life?

Not one iota of testimony in either of these directions can be adduced

in favor of preventive inoculation in rabies. Not one traveler, be he man or be he dog, horse, steer, or what not, that has once started on this terrible road of agony, has ever stopped short of that bourne from which nothing mortal has ever yet returned after once crossing its dread portals.

Show me that one, and I am willing to believe somewhat; show me a mild case that has been bitten and lived, where others have died, and again been bitten where still others have died of undoubted rabies, and then I will believe that preventive inoculation against rabies is possible. Until this can be demonstrated, I may be classed among the most skeptical of Thomases.

In support of the rabies or "hydrophobia" assertions of the owners of the cattle, as well as to show how good their case really appears to be, it must be admitted that in none of these cases in which the animals (and they include horses, cattle, swine, and dogs) have been said to have been bitten by a "mad dog," and have really become ill, has a single animal ever recovered.

This is certainly enough to differentiate this malady from any other with which we are acquainted, except rabies.

While the above discussion may seem somewhat out of place at this point, it seems to me that no where else could it be more appropriate.

Let us now return to our puppies!

I inoculated four of these with pure bouillon cultures, $\frac{1}{2}$ ccm. of the organism derived from the first Crete cattle, under the thin skin of the inside of one of the thighs. No local disturbances beyond a slight swelling and increased warmth occurred for a day or two. But what did result was most singular, and conformed in many respects, and I think most persons will agree with me, sufficiently with the phenomena seen in all the cattle. The course of the disease, as in the cattle, extended over eight to ten days, nine being the average in the cattle. The puppies became indisposed about the fifth day, but were neither excitable nor snappish, though very uneasy. They refused food and water, and *for two days, or so, before their death could not swallow.* Like the cattle, they would try to. The poor things did suffer terribly, their tongues hanging out of their mouths and becoming red and dry, the fauces and back parts of the tongue being covered with a viscid, glairy material. They also became severely constipated, passing semi-fluid fæces in small quantities in their strainings. They finally

became so paralyzed behind that they could only drag themselves or elevate themselves by the fore legs. Eventually they could do nothing. It must be especially mentioned that there was no paralysis of the lower jaw. It dropped, or hung, through weakness or distress; but they could always close it. At all times they showed pleasure at having their mouths wet with fresh water.

Diagnosis.—We now come to the bacterial results in Mr. Vance's steers killed November 8th. In this case were enjoyed many advantages which we did not have in the others at Crete, among the most important of them being the manual assistance of Drs. Bowhill and Thomas in making the autopsy, and also the cool weather. The material from which to obtain cultures was either removed as a whole, (the brain,) or in large pieces, and at once wrapped in napkins which had been soaking in five per cent carbolic acid solution, and then about half wrung out. This procedure is very practical for such field work as we have in Nebraska, where the trains are very inconvenient, the farmer's houses small, and to open tubes in the field very dangerous on account of the strong winds and possibility of pollution. By using the carbolized cloths, there is acid enough in them to kill any organism on the outside surface of the organs, while also forming a protecting coating for the inside of the pieces. On arrival at the laboratory a very large number of agar tubes were inoculated from different parts of the brain, the choroid plexus, the medulla oblongata, and the liver and the spleen. In most of these, pure cultures of one organism came to development in the tubes, which had been placed in the thermostat at 37.50° C., but only after the lapse of forty-eight hours—that is, during the third and fourth day. From one of these, bouillon cultures were also made.

Inoculation in Rats.—It so happened that I had a number of rats on hand, but no puppies, nor had I time to attend to the latter, as they would have had to be kept at the farm, some two miles distant, where I could not carefully overlook them, not having any calls in that direction at this time. Again, the rats seemed to be just the animals necessary, for the uninoculated ones could be turned into the others' cage, and given an opportunity to fight, if so inclined, the moment they showed indications of illnsss. The rats (four) were subjected to a sub-cutaneous inoculation of one-fourth of one cubic centimeter of a pure bouillon-culture under the thin skin of the inside of

the thigh, great care being taken not to touch the underlying muscles, the needle only being introduced sufficiently to penetrate the cutis. Naturally, they were etherized. They were also examined daily in the same manner for the first two days, during which time local disturbances of such a minor character as not to be worthy of mention were observed. On the third day they began to show signs of illness. They were very uneasy, and continually moved about, and refused food. On well rats being turned in with them, but watched, they tried to slink away from them, rather than to attack them, the well animal being the one which would have been aggressive had it had opportunity. This experiment was tried twice daily until the fifth day, when it was given up, as the inoculated animals had become too ill to be dangerous. Cerebral excitement of a serious nature failed entirely. On the contrary they could not swallow at all after the fifth day, though they would nibble food until the seventh. They could not drink, but would dip their mouths into water. Their backs were arched. They were extremely costive, and strained very severely, but the fæces, instead of being "balled," were pultaceous and very cohesive, being pressed out in small amounts at a time. Paralysis of the posterior extremities began on the sixth day, so that they could not rise on them, but would roll over on their hips, supporting themselves on their fore limbs. This condition of the posterior parts increased until it became almost total paralysis, and on the eighth day had extended to the anterior limbs, the animals lying on their sides and only raising their heads. So extreme was it behind that a hot wire applied to the toes of these limbs scarcely caused any reaction. Some was retained in the anterior limbs, and also in the skin of the abdomen. They retained control of their heads, however. All died during the ninth day *post inoculationis*.

From their organs the same germ was also obtained.

Description of the Germ.—This organism is not the easiest one in the world to handle, for it will not keep alive, extra-organismally, under artificial conditions of development but for a very few generations; and again, I have studied it but very imperfectly, as my duties on swine plague have been so exhaustive as to render it next to an impossibility for me to do very much detail work in other directions. Still, as this report shows, something has been done, and a large field of research opened for future investigators.

This object has a belted appearance, but is twice as long as that of the southern cattle plague, and again much narrower. It is navicular shaped, its pole-ends being decidedly pointed; these ends are not round, but longer than wide; the uncolored substance occupies two-thirds of the body when the object seems to have arrived at full development. I neglected to observe whether it is motile or not, or whether it would grow on the various media, simply because it was beyond my power to do so. I did study it somewhat on agar and in gelatine, however. On the former it grows as a yellowish-gray, (dirty yellow,) dryish, non-lustrous coating, which, as it becomes aged, is very friable. Cultures must be changed just as soon as they arrive at a good development, or they will refuse to grow. Coccoid degeneration occurs rapidly; in fact, so fast that in a three-day-old-culture it is often difficult to obtain normal specimens. How long the thing will keep its virulence I do not know. In bouillon it develops better than elsewhere, being mostly anaerobic, but sometimes forms a delicate patch of film on the surface, which at once disappears on shaking the flask. In gelatine it develops, and that is about all; and then, seemingly, though I say it with reserve, only at a temperature on the extreme limits of fluidification, the thermostat offering more favorable conditions. The germ does not fluidify gelatine. It grows as individual colonies along the line of culture, but more individual than either swine plague or the other organisms described in my work. It is aereobic to a degree, and, on the gelatine, at the point of puncture it may develop characteristically; but that can only be asserted when it is positively known that no other organism develops in the same way. It does not extend over the surface to any marked extent, but rises up from the point of puncture as a sort of verrucous growth, the material being a dry, friable, crust of a yellowish-gray color, with a granulous surface. In fact, its character can be compared to the appearance of a dry, caseous material forced out of a small opening by pressure. It cannot be carried along in gelatine cultures, at room temperature, for but two or three rapidly repeated generations. I have not the time to give to the detailed study of the histological lesions in the organs of the cattle and inoculated animals, but the competent pathologist can easily read the chief microscopic changes in the macroscopic descriptions previously given. The organism has, however, been carefully traced through all these tissues, and appears to

prevail largely in the smallest capillaries, though in can be seen outside of them. It is exceedingly difficult to demonstrate, but it can be done. The smear-covering-glass method recommended in the discussion of the germ of the yellow fever can be most satisfactorily applied here. One striking lesion seen, even in slides colored to demonstrate the germs in sections of the brain, is the amount of red-blood cells in the lymph-sheaths, and the great number of leucocytes present in the same, the blood vessels also being very much engorged. The parenchymatous changes in the glandular organs are generally of a minor character, being those of a clouded swelling. Extreme degrees of fatty degeneration are rarely present.

DIAGNOSIS.

What is this disease in cattle which we have been studying? That is a question which will be left open for future investigations to decide, though we shall take pains to demonstrate how completely this "hydrophobia" fits into the picture given of rabies in some of our principal text-books, in its clinical phenomena, leaving it to later investigators to show its correspondence in some essentials with the results obtained by investigators in the experimental study of rabies.

That this "hydrophobia" as observed in cattle is an idiopathic malady would seem sufficiently demonstrated to go beyond question. That in all the cases personally investigated there has been a dog in the story, and that the owners, and others, stated the dog to have been "mad," has been sufficiently shown. That the disease, or something leading owners to report "hydrophobia" in their live-stock, occurs at all seasons of the year, and in almost all classes of domestic animals, has been illustrated by the few quotations from news and stock papers which have come in our way. It may be well to state that the number of these reports could have been greatly augmented had pains been taken to collect them as they occurred; but it was not my intention to report upon this disease at present. But my final leaving Nebraska, and my desire to give all the time possible to the study of the non-recurrent diseases of child life in the future, rather to than those of animals, has made it imperative that even these unsatisfactory and incomplete investigations be published in order that they may serve to guide other workers in this all-important field of national economy and public health.

The idiopathy of this "hydrophobic" disease is well shown in its duration, its average course as far as studied having been almost invariably nine days, both in the cattle and the puppies and rats which were inoculated. This in itself is sufficient to differentiate it from any other known disease of cattle. This idiopathy is, however, still more pregnant when we come to consider the clinical and pathological phenomena in this disease. The former clearly show that we have nothing before us belonging to a disease of a strictly hæmic character such as the southern cattle plague, or the corn-stalk disease, anthrax, or black-leg. We miss the specific lesions of the latter in the muscles, the enlarged spleen, gelatinous infiltrations, and black, tar-like blood of anthrax, but especially the unmistakable *B. anthracis*, as well as the severe parenchymatous complications and intestinal complications of all these other diseases. The hæmaturia so common in the southern cattle plague is wanting, as well as the presence of the Texas cattle to cause it. Its occurrence at all seasons of the year again shows the impossibility of its being the corn-stalk disease; but beyond that the extreme scarcity of its germ in the blood, and the great difficulty of obtaining developments of the micro-organism found, and its very short existence under artificial conditions, all point to a disease having no connection with any other disease known to us in this part of the country, not excepting the "mad-itch," of which I know nothing. In this connection it may be well to mention that none of these animals showed a tendency to bite or gnaw their own bodies.

On the other hand we have seen, aside from its constancy in duration, that in this disease there is invariably a developing paralysis of the digestive tract manifested by inability to swallow after the third or fourth day, and extreme constipation and tenesmus, as well as approaching and finally complete paralysis of the limbs, generally more extreme in the posterior than the anterior, showing lameness of the motor centers. That other nervous centers are complicated is evinced by the amaurotic condition of the eyes, the frequent micturition, and the oestrum, under unnatural conditions, in the cows. Another most striking point of differentiation from all other diseases is the invariably fatal termination of this so-called "hydrophobia."

RESEMBLANCE TO RABIES, AS SHOWN BY QUOTATIONS FROM STANDARD AUTHORS.

Spinola, (Handbuch der Spec. Pathologie Therapie, 1858, p. 1555,) says:

"Cattle, when bitten by rabid dogs, are more frequently subject to the disease than horses. The phenomena observed are dependent upon the same organic disturbances as in other animals, though the symptoms vary somewhat in different cases.

"The disease begins with loss of appetite, depression, and irritability and uneasiness; on being disturbed, the animal at once becomes more uneasy and furious, eyes fixed, pupils distended, saliva and froth frequently from the mouth, bellow much, and with a hoarse, changed voice; stamp with fore feet and easily become excited on the presence of strangers, or other animals; tendency to bite some one. Constipation at first, later on diarrhœa; tenesmus constant. Urinate frequently in small quantities. Sometimes all these phenomena are present in one individual; at others some may be wanting. Paroxysms of fury, varied by periods of exhaustion, especially on irritation; soon become emaciated; paralytic phenomena are frequent. Sexual irritation frequent."

Roell, (Pathologie and Therapie d'Hausthiere,) says:

"The phenomena in cattle are essentially the same as those seen in horses—depression, uneasiness, great irritability, muscular spasms, foaming at the mouth, sexual irritation, difficulty in swallowing, and irritation at the locus traumatica.

"During paroxysms eyes become distended and reddened, fixed, dilated pupils; voice changed to a peculiar hoarse, dull sound, which is frequently emitted; stamp with fore feet, often fall to the ground, but soon rise again; seek to become free from attachments when fastened; strike with horns, and often attack other animals; appetite and urination soon cease entirely; excrements at first hard, finally become soft; tenesmus constant; urinate frequently, and in small quantities; become emaciated, and finally fall into paralytic and soporous condition."

It is not my intention to offer any other evidence in favor of this cattle "hydrophobia" which has been reported on here, being virtually rabies, than the above descriptions, which will be found to correspond with those given in other text-books.

Any one, however, who will take pains to carefully review the experimental studies of rabies which have been made by European investigators during the last few years, especially a recent publication

by Di Vestea and Zagari,* will see how very closely the results in their inoculated animals correspond with those obtained by me in the puppies and rats. Whether or not the micro-organism used in my experiments be really that of rabies; whether, which seems impossible, further investigations should show that the disease in the cattle is really the rabies, and this germ a secondary or accidental pollution, still the fact cannot be escaped that the results obtained were not only very astonishing, but that they do bear the strongest possible resemblance to those obtained by experimentors in inoculations with what seems to have been determined to be actual rabies virus, even though no specific germ has been discovered in it.

I have purposely neglected to speak of the bacteriological results obtained from the cow at Dorchester. Suffice it to say, that so far as a microscopical examination and cultures can determine the point, the same micro-organism was derived from this animal. I could not possibly find time—nor did I have conveniences then, everything being occupied in experiments on swine plague—to make any inoculative tests.

Before closing I must refer to another outbreak of disease in cattle, which bore the strongest resemblance to "hydrophobia," but in which there is no satisfactory evidence of a reputed "mad dog" having been present.

CATTLE DISEASE AT GRAFTON, NEBRASKA, JULY AND AUGUST, 1888.

"GRAFTON, NEB., July 17, 1888.

"*F. S. Billings, Esq., Lincoln:* MY DEAR SIR—Yours just on hand, and in reply will say that we did not let the cattle die their natural death, as the three were shot. But the one you have seen took sick on Friday, and you shot it the next Friday. You said at the time that the heifer might live twenty-four hours more. My opinion is they will live seven or eight days. The first notice which I have observed—I lost three—they stand still about one-half of the time in twenty-four hours; then they begin to bellow day and night, most all the time, and act as though they were mad, with saliva lining their mouths. At first notice you can scare them off by making a noise; but the longer they are let go, the more vicious they become. Also, they are more vicious as they are older. They will always follow their mother, and seem all the time they would like to eat and drink, but

* "Fortschritte der Medicin," Bd. 7, 1889, Nos. 7 and 8.

could not swallow. I was of the opinion the disease affected the brain. There seems to be no more sick among my cattle. The three sick would chase the younger cattle all around the pasture, and would suddenly stop and chase the little birds until they would fly. The second one would bite the shrubbery which grows in the pasture.

"Yours respectfully, JOHN W. MCKELVEY."

In a second letter, in answer to some questions, Mr. McKelvey says:

"You say something about a long interval between each case, if I understand you right. The first one was in April; thirty-eight days after, the second occurred; and thirty-two days after that, the third and last one."

From these letters the exact resemblance to the "hydrophobia" cases can be seen, the duration of the disease, "seven or eight days," being about the same, the inability to swallow, the cerebral excitement, and constant bellowing, all point to the same disease.

PERSONAL OBSERVATIONS ON MR. MCKELVEY'S THIRD ANIMAL.

July 12, 1888. Red steer much emaciated; conjunctivæ injected; pupils of eyes distended, but animal could see. Upon the least disturbance, the steer would commence to bellow in a rapid, hoarse, and unnatural manner. It did not display any desire to attack us, and being very securely fastened, it was possible to take its temperature and pulse. Former 40° C., latter 115, and weak; respiration rapid, but not labored; marked rectal tenesmus, with partial eversion of the mucosa on straining, when it passed a frothy slime, with small quantities of semi-fluid fæces. Micturition very frequent, and in small quantities. Could not swallow, but would take the food and water into its mouth as described in the other cattle; would tip the bucket over, but did not get "mad" because it could not swallow.

Animal shot through the heart.

Necroscopy.—This was very superficial, the brain not being removed, as it was very late in the evening, and we wanted to get a train, which we missed; and having no material to make cultures, they were lost in this case, though alcoholic specimens were preserved.

Blood of purple-red color, somewhat thick, but oxidized rapidly on exposure to the air. No effusion in cavities of the body. Lymph-glands much swollen, of a pinkish color, and very juicy. *Kidneys,*

swollen; cortical substance gray-red in color, opaque, and anæmic; medullary bright red. *Liver*, swollen, cut surface yellowish-gray-red in color; opaque and anæmic; acini distended; peripheries yellowish-red; central portion red. *Spleen*, neither swollen nor juicy. *Lungs*, normal. First two stomachs apparently normal; third, hard, partially contracted, contents abnormally dry; fourth, quite empty, mucosa swollen, and of a diffuse bright-red color, interrupted by a few circumscribed darker red spots of various dimensions. Small intestine partially empty; contents quite fluid; mucosa of anterior portion swollen and diffuse red, while in ileum it was marked by diffuse patches of capillary engorgement. Contents of large intestine semi-fluid; mucosa as in ileum.

As no cultures were obtained, the examination of the alcohol material was postponed until a recent date, when apparently the same organism was found both in sections and in alcoholic-smear preparations as in the same materials from the Crete cattle and Dorchester cow.

Capt. Real's Calf.—On August 13th I visited a calf belonging to a Capt. Real, of the same place, which displayed the same symptoms as the above steer, both clinically and necroscopically. One, which seemed to attract the captain's attention more than any other, was the marked straining of the animal, and eversion of the rectum at the same time. To use his expression, the "bum gut turned itself inside out." I tried to obtain cultures from this animal, but every tube remained sterile, which at least speaks for successful manipulation in an open field. Examinations of the tissues, however, revealed apparently the same organism as in the other cases.

Capt. Real denied the presence of a mad dog most positively, while Mr. McKelvey reported that a suspicious one was in the vicinity of his place "about one year ago." These statements have no value pro or con, as it would be possible for such an animal to pass through one of our western pastures unobserved, and still bite some of the cattle.

With this I close the story, leaving it to future work or other investigators to endeavor to definitely settle the question of the etiology of this very singular malady, and the positive connection, if any, of this peculiar micro-organism with it, by direct experiments in cattle, as well as further experiments in dogs. Before closing, however, I would call the attention of my American confrères to two statements

which may escape their notice, being only to be found in foreign literature, but which seem to have been established by European experimenters, viz.: in inoculation experimentation in rabies —

1. Intra-ocular injection of suspected or actual infectious material has been found to be equally trustworthy to the much more troublesome intra-cranial method.*

2. It suffices to cut the skin and open up a fibre or nerve and lift it on the point of the knife. Then cut it in two and pour a drop of the virus into the wound, so it surely comes in contact with the cut end.

This has been found equally reliable to injecting the virus directly into the substance of a large nerve. To introduce it into the sheath of a nerve has been found an unreliable procedure.

PREVENTION.

From all which has been said, and from the numerous reports in the newspapers, and the general experience of our farmers, it must be admitted that there is quite a prevalent disease among our live-stock, in some way connected with the presence of dogs which are asserted to have been "mad." That fact must remain undisputed whether it is admitted that I have either been studying that disease or even found its germ.

The question then is, *what are we to do with the dogs?*

Some inconsiderate persons might say, "kill them all off;" but the writer is not one of that kind. Turn where we may, the truest picture of unselfish and absolute devotion which we ever see is given by the dog. The brotherly or humanitarian spirit to one another in our own species sinks into insignificance in comparison with that exemplified by the faithfulness of the dog to those to whom it is attached. Curs are no exception to the rule. The legends of Indian mythology tell us of a certain prince who gained the rewards of eternal happiness because he did not desert his dog, the form of which a god had assumed, "the god appearing sometimes thus" in his journey to the heaven of the just. On the other hand, it must be admitted that in some unknown way, even this truest of friends sometimes carries with it the stings of a death so horrible that language fails in its ability to picture it. It is true that there are too many dogs; too many

*Di Vesta and Zagari, l. c.

irresponsible canine curs, as there are too many human beings who seem to have been born but to be a curse to their species; too many dogs without any one to be responsible for their actions. It is self-evident, then, that some legal steps must be taken to lessen their number. They cannot be confined in asylums or jails as we do with irresponsible human beings.

The first step necessary is to lessen the number of females. This can only be done by placing a high license upon them. To the writer's mind, no one should be allowed to keep a "bitch," unless he paid a license of twenty-five dollars, except the breeders of fancy dogs, who should pay a special license, to be known as a "breeder's license," of not less than one hundred dollars a year. Male dogs should be taxed five dollars each. But a special favor should be shown to "spayed" bitches, which should be placed on a level with male dogs, as they are even less dangerous than that class. All licensed dogs should be recorded, and the record kept in the office of the local police. They should be required to wear an official collar with the name of the owner and number of the license upon the same. All dogs not having such a collar should be peremptorily shot wherever met. In no other way can the dangers of rabies be limited to the lowest possible degree, and these losses in our live-stock be brought within the minimum limits.

I must say that I deeply regret my utter inability to have given the time to the experimental study of this disease which its importance and mysterious character certainly demand; but having done all I could, my simple duty of recording the results obtained has been fulfilled.

ARTICLES IV. AND V.

ARTICLE IV.—CONTAGIOUS INFLAMMATION OF THE CORNEA IN CATTLE.

ARTICLE V.—A SINGULAR DISEASE OF THE SEXUAL ORGANS IN COWS.

ARTICLE IV.—*Contagious Inflammation of the Cornea in Cattle.*

KERATITIS CONTAGIOSA IN CATTLE.

This is not a new disease by any means, so far as the United States are concerned. Nevertheless I have been unable to find any description of it in the literature at my command. While new to myself until the past summer, there have been quite a number of reports of its existence, and complaints about it, from farmers and breeders of cattle in some of the live-stock journals of our Western States. Under these circumstances, it would seem that a description of its clinical phenomena and gross pathological lesions may not be without scientific interest to the opthalmologist, and have some practical value as well, especially as experience has shown that the extension of the disease over the members of a herd of cattle can be easily prevented by isolation measures, and its course much shortened by the mildest and simplest therapeutic treatment.

History.—During the past year, three quite extensive outbreaks have been reported to me in Nebraska; one having been at Kearney, and another at Gibbon, in Buffalo county, while a third occurred in the immediate vicinity of Lincoln, thus giving an opportunity for some personal observations. Of the outbreak at Kearney, the owner wrote me that "the trouble appears to begin as a small spot on the eyeball, the eye running and gradually growing worse, showing a purplish color, and becoming very sore. The pupil seems to protrude as though proud-flesh, or something of that nature, grew in the center." The above will be found to be an unusually good description from the hands of a layman.

The disease first appeared, in the vicinity of Lincoln, in a herd of dairy cows, about July 1, 1888. Its extension over the herd was very slow indeed, and although there were several horses among the cattle, exposed to more or less danger of infection, the disease did not extend to them, nor to the men milking and caring for the animals. Up to October 1st, ten cows, out of about seventy-five, and seventeen calves,

had become affected, at which time my observations had to cease on account of important engagements elsewhere.

Clinical Phenomena and Gross Pathological Lesions.—The disease first manifests itself by the discharge of a thin, clear, watery fluid from the conjunctival sac. Marked photophobia is an early symptom, the eyelids being closed and somewhat swollen, though the afflicted animal can open them easily enough if startled, and has complete control over their movements. The discharge rapidly increases in quantity, the conjunctiva becoming more and more swollen, until, in severe cases, the engorgement of the vessels becomes so intense that its general color is almost a diffuse copper-red. (This has not been shown in the accompanying illustrations, on account of the unfortunate necessity of all possible economy in the number of cuts.) In many severe cases, the discharge becomes purulent. While a careful examination of the diseased animals has shown that the rise in temperature is but very slight, still they present phenomena which the casual observer might mistake for those of high fever. Their heads are held depressed, the ears becomeing pendulent. They refuse to eat, and rapidly emaciate, while the yield of milk lessens materially. These conditions augment during the first eight or ten days, the photophobia correspondingly increasing; instead of to fever they must be attributed to the severe pain which the animal is suffering. Intra-ocular pressure is present in an excessive degree, but the cause thereof is entirely to be sought in a marked increase in the quantity of fluid in the anterior chamber of the eye, the cornea of which becomes distended and very prominent. While the disease has never been reported to me as beginning in both eyes at one and the same time, still, in almost all cases, it has been noticed to extend to the other eye.

At about the second or third day from the time the first eye has been observed to be affected, a very delicate cloudiness makes its appearance at or near the center of the eye, which continually increases, the membrane becoming thicker and thicker, and more and more opaque, which conditions gradually extend to the sclerotic edges. This center is at first of a pearly-white color, and in some cases may not become more than a creamy-white, being much thickened, but in many a small yellowish speck will be seen to form, which gradually becomes larger, the tissues over it becoming thinner and thinner. This yellowish spot will be seen to become surrounded by a wall of

thick, swollen, indurated tissue of a white color, while outside of this will be a more or less pearly-white substance, losing itself in a bluish-white tissue as the peripheries of the cornea are approached. [See Fig. 1, plate IX.] (It will be self-evident that all these fine points of shading could not be illustrated to perfection except at great expense; hence the reader must make due allowance for illustrations in which it has only been endeavored to show the essantial points.) From this indurated tissue, which encloses the apostematous center, may be seen numerous delicate blood-vessels, taking their course in a serpentine manner toward the sclerotic edges of the cornea. This vascularization is often so intense, as well as so delicate, in the character of the neoplastic vessels, as to give to the tissues a diffuse, dark-red color, even to the degree of hiding the larger vessels from sight. In fact, so extreme may this become, that it will take careful observation not to be misled in assuming the existence of an excessive intra-corneal hemorrhage, completely filling the anterior chamber of the eye. *The latter never occurs, however.* As the processes increase in intensity, the yellowish center increases in extent, and always in a direction across the eye, from side to side, and then below the same will be seen a mass of intensely vascularized tissue, much swollen; but still the overlaying tissues will retain their normal lustre, or nearly so, while those over the yellowish center have become very thin and lustreless. [Fig. 2, plate IX.] In many cases these abcesses rupture, and the contents at once escape. The rupture is invariably of the external tissues at first, the augmented amount of fluid in the anterior chamber exercising a perfectly equal pressure against Decemet's membrane, preventing a rupture in that direction. But when the abcesses have been unusually extensive, the tissues forming its inner wall are too thin to resist this pressure, and a rupture soon follows, with escape of the aqueous humor, and prolapse of the lense, followed by the utter destruction of the complicated organ. In the majority of cases this fatal termination does not occur, but from the ruptured edges of the external walls and base of such an abscess, the development of granulation tissue begins, and extends across the cavity, completely filling it up, and projecting to a considerable degree beyond the level of the surrounding tissues; naturally, the size and shape of this mass will vary with the size and shape of the original abscess, and extent of the rupture. Such conditions have been illustrated in Figs. 3 and 4,

plate IX. From these granulations the previously-mentioned vascularization may be seen extending in all directions toward the peripheries of the cornea. To one observing this disease for the first time, and before a complete examination of a severely-complicated eye has been made, the most natural hypothesis would be, that there must also be very extreme complications of the internal portions of the organ. He would find himself severely mistaken, however. *Aside form an œdematous condition of the iris, the internal portions of the eye remain absolutely normal.* Even the aqueous humor, while increased in quantity, remains as clear and pellucid as the clearest of distilled water, as I have tested in every case by the careful withdrawing of the same with a sterilized glass-barrelled syringe. The most exact microscopical examination has failed to reveal the presence of a single leucocyte.

Microscopic Examination of a Flat Section of a Diseased Cornea.—Examined microscopically, the first thing that strikes the eye is the immense number of newly developed blood vessels in and extending from the specially diseased portion of the cornea in every direction, with apparent anastomses between them, towards the peripheries. In the peripheral portions numerous round and oblong nuclei can be seen, as well as round cells, with one or more nuclei in their bodies. The latter are often collected in immense numbers, especially in the vicinity of the blood vessels, while the former, in many places, present a picture more or less resembling that of connective tissue. Toward the center of the diseased portions of the cornea, the blood vessels are much larger than in the peripheral parts, while the round cell infiltration is very dense; but between the round cells can be seen many elongated cells having a granulous body, as well as the before described round and elongated nuclei. In certain places the accumulations of round cells are so dense that one can scarcely distinguish the individuals. An occasional granulous body, and sometimes clusters of the same, may be seen in the diseased tissues.

A Micro-Organism in the Above Section.—In sections of the cornea colored in a corbol-fuchsin-methylyn-blau, and Beck's double stain, may be seen a short, thin bacillus, with round ends, and in which is to be seen either a clear center or a spore. Sometimes the short body seems divided by a coccus-like dark spot in its center. Such an organism may either represent two organisms, with a spore in each, or

it may have a belted appearance. On the other hand, innumerable individuals may be seen presenting directly coccoid ends and an uncolored dividing portion. In other cases objects of exactly the same dimensions in all directions are present, but colored diffusely throughout. Dense clusters of micro-organisms presenting all the phases already described, are to be seen in various parts of these sections. As has been already mentioned, all attempts at transmitting this disease from diseased to healthy cattle proved negative; hence, though we obtained the above-described germ in pure cultures, but did not experiment with it on account of being called away, and especially on account of the failures in direct transmission from the eyes of diseased to healthy cattle, I prefer to say nothing more about it, thus leaving the field open to others for future investigation.

Termination.—Singularly to say, notwithstanding the apparent severity of the external lesions, with the exception of the rare cases in which complete rupture of the cornea with prolapsus of the lense occurs, there is, or, better perhaps, has been, an absolute return to normality, and complete re-acquisition of sight. Where no rupture has occurred, or where there has been no accumulation of pus, the first step toward restoration is a decrease in the caliber and number of neoplastic vessels, with a clearance of the cornea at its peripheries, which slowly but surely extends toward the centers until the entire organ is again as transparent as could be desired. Where there has been a rupture, and the cavity caused thereby filled by granulation tissue, the same processes occur, and the same phenomena are seen, the vascularization decreasing and the granulation tissue becoming more and more anæmic, and less and less prominent, until it finally entirely disappears, its place being at first represented by a yellowish-white, then white, and finally opaque, pearly spot, which eventually disappears entirely, the external epithelium having again completely covered the spot, and restitution is perfected in a manner seldom, if ever, seen in any process of wound-healing where the previous lesions have been of such apparent severity.

Nature.—The extremely slow manner in which this disease extends over a herd of cattle, has been previously mentioned. No less remarkable has been the (in my experiments) absolute impossibility of intentional transmission of the disease from afflicted to healthy animals. For example, completely sterilized plugs of absorbent cotton

were placed in the conjunctival sac (of a bull recently affected, but with a profuse discharge,) until completely saturated. In one case, such plugs were placed within the same sac of the eyes of a healthy calf, while in another the cornea had been previously scarified with a sterilized lancet. Notwithstanding the lids were held closed for five minutes in each case by attendants, *the results were absolutely negative.* The aqueous humor of the eye of a diseased calf having been withdrawn with a sterilized syringe, was injected into the anterior chamber of the eyes of a rabbit, and, as well as possible, into the tissues of the cornea of another, with the same unsatisfactory results. They do not, however, have any value of evidence against the conclusion that the disease is of a contagious character. For this speaks its appearance in a single individual at first and its very gradual extension; while were it due to any specific, external cause, where all the animals are banded together, and hence received the same treatment, a more general eruption would certainly occur.

Prevention and Treatment.—Complete isolation of the diseased from the healthy animals will cut off the extension of an outbreak, with the most happy results.

All the treatment necessary is a dark place, and cloths constantly hanging over the eyes, kept wet with cold water all the time. This treatment lessens the severity of the disturbances, and hence tends to shorten the period of restitution.

The application of any washes or remedies inside the conjunctival sac is not only useless but harmful, as the animals resist it all they can, and hence the danger of the introduction of irritating foreign material is increased, and even the endeavor to lift the lids, or handle the eye, must be looked upon as having an injurious tendency.

Article V.—*A Singular Disease of the External Sexual Organs in Cows.*

A Singular Cattle Disease.

August 16, 1888, word was received at the laboratory that great alarm was being felt among cattle owners in the vicinity of the town of Shickley, Nebraska, on account of a hitherto unseen disease in a herd of cattle, which had extended to a great many individuals. Notwithstanding most pressing engagements in other directions, this outbreak was visited. Upon inquiry, the trouble was found to exist in but one herd, which was quite large, consisting of 293 animals, two-thirds of which were cows and heifers, the remainder being steers and a few bulls. The cattle were herded on an open prairie by cowboys, and corralled every night. The disease was found to be absolutely confined to the females of the herd, and extended to the sucking heifers as well as old cows. It is to be regretted that it was impossible to photograph some individuals which presented the most marked lesions. The disease commenced with tumefaction of the fleshy parts of the vulva, which became very hot. Then small, hard nodules would develop, which at first were sharply circumscribed, but eventually coalesced. These nodules soon presented a broken surface, which became red and granulous, like proud flesh, until the whole vulva would become complicated in this way, with islands of intact skin in places. With the exception that there did not seem to be any secretion from the granulous surface, or any formation of abscesses, or disintegration of tissues on or beneath the surface, the whole disturbance presented much resemblance to carcinomatous infiltration. The malady did not seem to be necessarily fatal, as many cases of auto-healing were reported, and some shown; but when healing did take place, or better, where I saw cases of such, there had been extensive formation of cicatricial tissue, which led to the most deforming retraction of the parts. This process often led to the death of the animal, in that the meatus of the urethra became involved and completely closed, so that the animal died in intense agony from reten-

tion of the urine, and the natural renal and constitutional complications which necessarily result in such a case. In one animal of this kind the meatus had become so complicated and constricted that the urine was passed in a stream no larger than the ordinary steel knitting needle, and with so much force as to be squirted several feet from the body.

The primary origin of the disease is buried in absolute obscurity; but its outbreak in this herd was reported to be due to an old white cow that was present, and which was said to have been known to have been afflicted for the past four years. This being so, it shows that a very chronic course is possible. This cow was very much emaciated. The external sexual organs and anus were much deformed and drawn in, but the *meatus urethralis* was free. Ulceration was still present. Healed portions could be seen, but the attendant cow-boys reported that new eruptions were continually occurring. The anus was also constricted, and the animal defecated with some difficulty. The diseased parts presented a very disagreeable appearance, being covered with a dirty black escarous mass, interrupted by protruding patches of red granulation tissue, which bled somewhat from the friction of the tail. The external lymph-glands of the animal in the vicinity of the udder were enlarged and very hard, but those of the anterior parts of the body did not seem to be affected. I tried very hard to purchase this cow and a freshly-diseased calf; but for the first time in my experience in Nebraska, the owner wanted a most exorbitant price—in fact, was unapproachable—simply because he thought I would have it at any price. In this he was mistaken. I was allowed to snip off a small piece of the granulation from a calf, however, and tried to induce the disease by rubbing the same into scarifications made in the skin of the vulva of a healthy heifer at the station. The results were entirely negative. I also inoculated a female rabbit with a small portion of the same material by scarifying the lining of the vulva. The animal died of septicæmia, due to a small bacillus, the further history of which was not followed. In this outbreak this disease is strictly limited to females. The cow-boys positively asserted that they had seen the bulls mount diseased females, and that not one of them derived any harm from it. Certain it is that no evidence of the same was to be seen. The introduction of the disease by a single cow into this herd, (made up of a large number of cattle belonging to an

equal number of owners, which had never been thus mixed until brought together on this pasture in May, 1888,) and its gradual extension to nearly every female of the lot, two-thirds of which were of that sex, certainly points strongly to a contagious disease; for were there a common cause present, the males should have presented lesions in some way. On the other hand, the manner by which it was extended seems to be very mysterious, there being only one way open to reason, and that by the animals licking these parts, and thus conveying it to each other. But this hypothesis seems to be more or less contradicted by the negative results following any attmept at transmission, which, however, had not much value; for had it been possible to obtain the desired diseased animals, or had the herd been where it could be visited at pleasure, and not over one hundred miles away, other and more successful results might possibly have been attained.

This has not been the only outbreak of this singular disease in Nebraska, however. It was subsequently discovered that a considerable eruption had occurred in the vicinity of Kearney in the winter of 1886, and caused great alarm, many animals having been killed. In this outbreak steers were also affected, the disease attacking the tissues around the anus. From a Mr. Rogers, of Gibbon, it was learned that the disease could be cured by treatment, a discovery which he accidentally made in the following manner: He ordered his son to turn two very badly diseased steers out in a field, and to let them live or die, as it might result. The son thought he would experiment a little, and procured some pure carbolic acid, with which he completely saturated the diseased parts, and in no very careful manner encroached upon the tissues embracing them. The natural result, a severe sloughing, followed, but the animals recovered very rapidly, this severe treatment fortunately not being followed by any obstructing interference with the natural passages. While it is to be hoped that this is the last of so damaging and alarming a malady, and that it is not contagious, but due to some other cause, still it is equally desirable, should it occur again, here or in other places, that other investigators may be more fortunate in discovering its definite nature and etiology by finding more accommodating and rational owners, which, to the credit of Western farmers be it said, is likely to occur in most cases.

In the words of Georg Simon Winters, in his wonderful "Ross Artzney Kunst," 1678, I will now close my work in Nebraska:

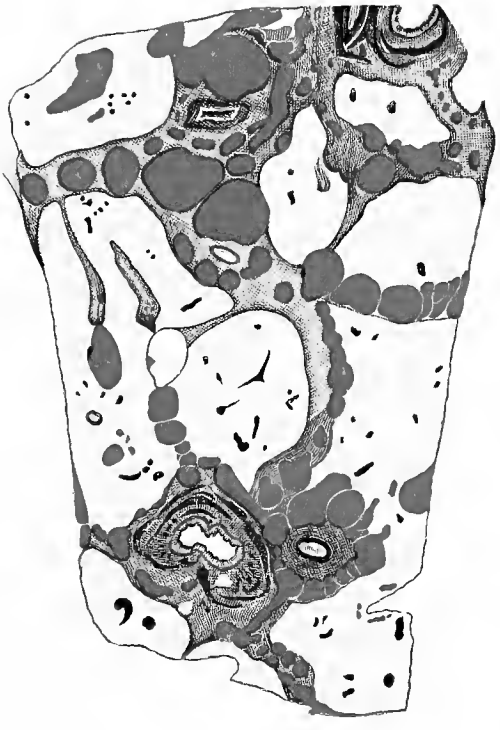
"Und dieses ists, was ich nicht allein mit grossem Fleiss und Mühe aus guten und bewahrten Autoren zusammen getragen, son dern auch in meine ernsthafte Studien selbst gut und probat befunden habe. Will also für dieses Mal beschliessen und sagen, Ein Anderer machs besser."

"Ein Anderer machs besser." As this ancient author closed his work with the wish that another might be able to do better than he had, so I hope that my successor may exceed me in diligence and honest service to the live-stock interests of Nebraska; and thanking those who have so nobly supported me in my work, I now leave it in the hands of the stock men of Nebraska. THE AUTHOR.

Lincoln, Neb., May 24, 1889.

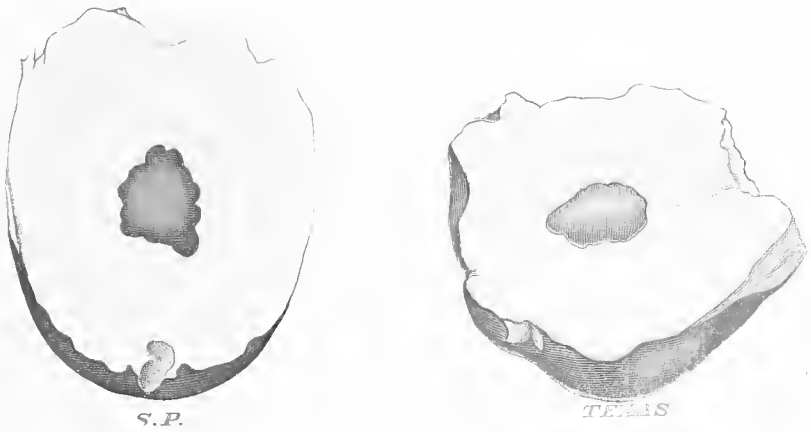
INDEX.

PLATE I.



Coagulation obstruction of circulation in the lungs, leading to necrotic pneumonia, as seen in the Corn-Stalk Disease and Swine Plague.

PLATE II.



Growth of the Germs of Swine Plague, the Corn-Stalk Disease, and Southern Cattle Plague on Potatoes.

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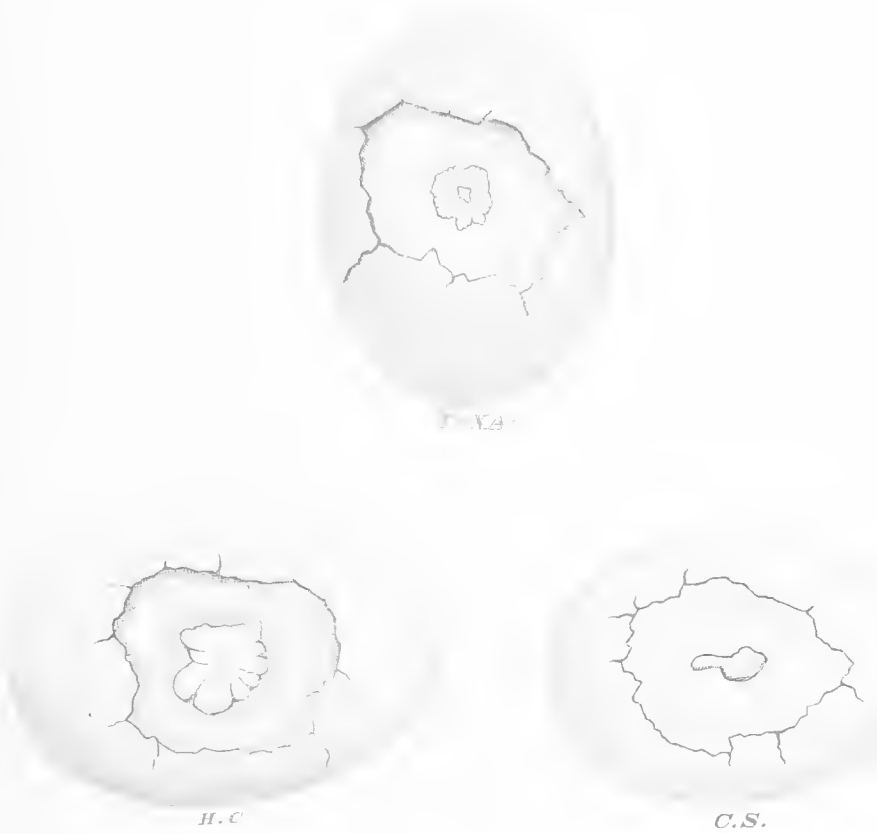
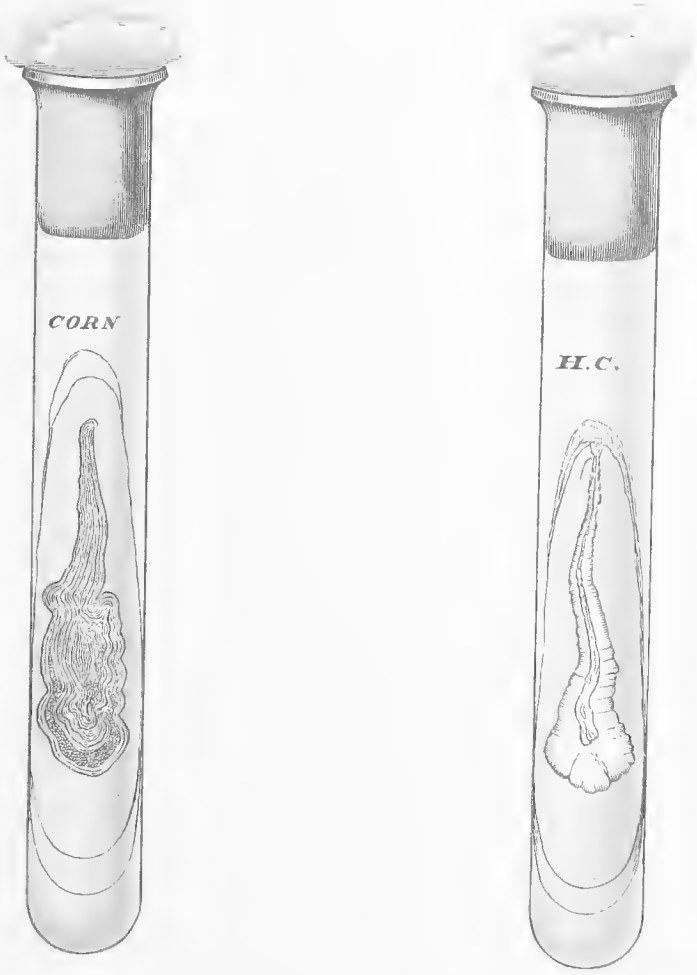
PLATE III



Growth of the Swine Plague, Corn-Stalk Disease, and Southern Cattle Plague Germs on White of Eggs.

PLATE IV.



The Corn-Stalk Disease Germs and those of Swine Plague,
as they develop upon Agar-agar.

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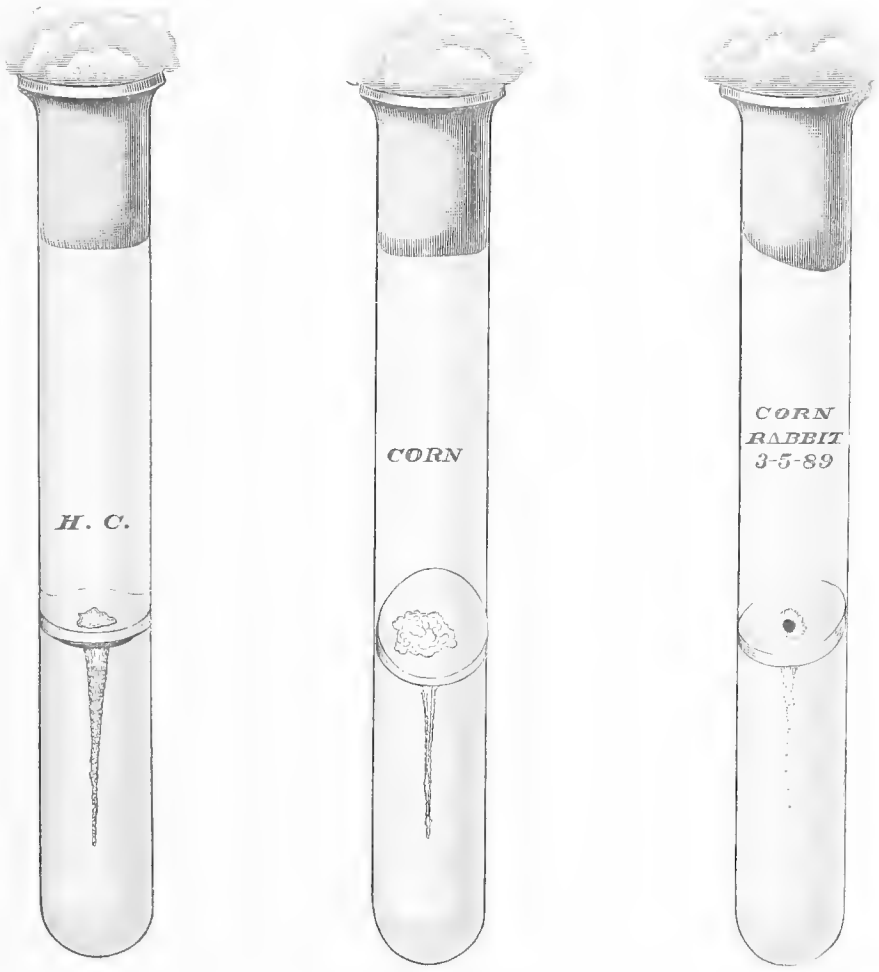
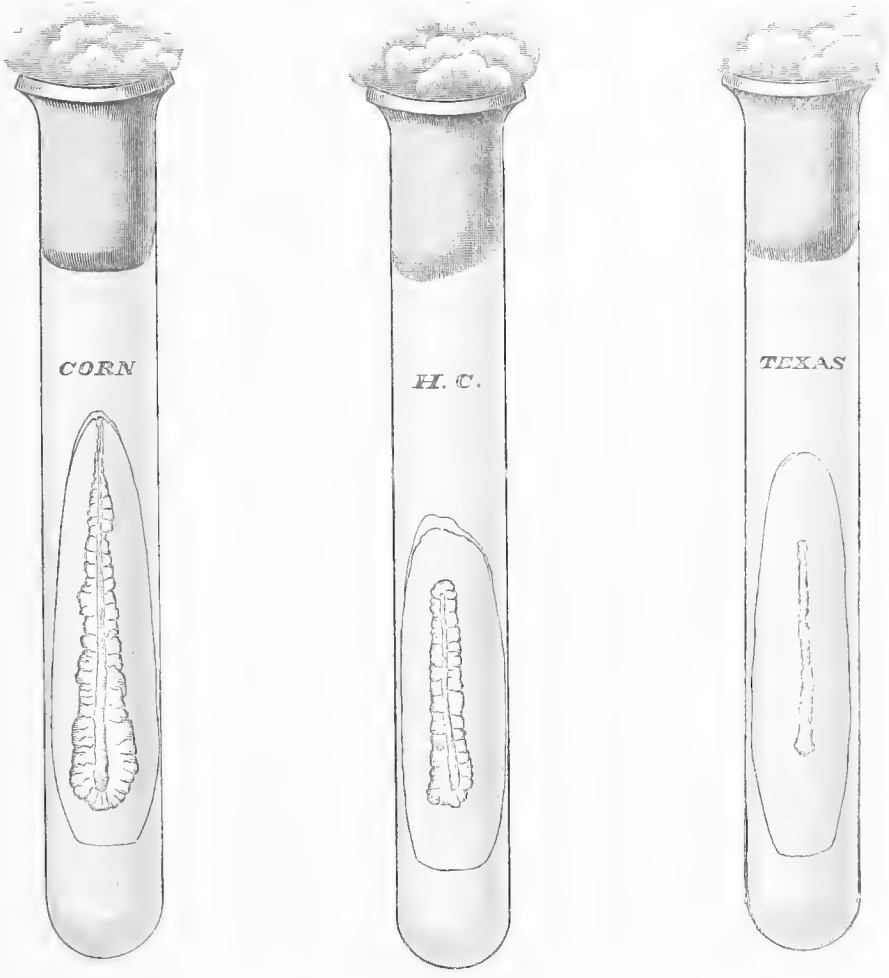
PLATE V.



Puncture developments of the Germs of Swine Plague and the Corn-Stalk Disease in gelatine, the third tube being a fresh culture made from the blood of a rabbit having the Corn-Stalk Septicæmia.

PLATE VI.



Cultures of the Germs of the Corn-Stalk Disease, Swine Plague, and the Southern Cattle Plague, upon the oblique surface of gelatine, all being of the same age, showing the variation in manner and rapidity of development.

PLATE VII.

Diffuse capillary engorgement of the Kidney as seen in the Southern Cattle Plague.
From the U. S. Agr. Report 1871.

PLATE VIII.

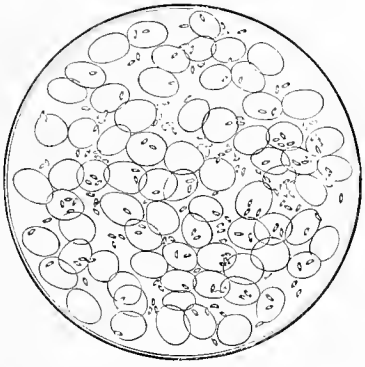


FIG. 1.

Mature form of the Germs of Swine Plague Southern Cattle Plague, Yellow Fever, and the Corn-Stalk Disease as they appear in specimens made from the blood, or in smear preparations from the tissues.

FIG. 2.

Photograph of a specimen of the Swine Plague Germ according to Dr. Detmers.

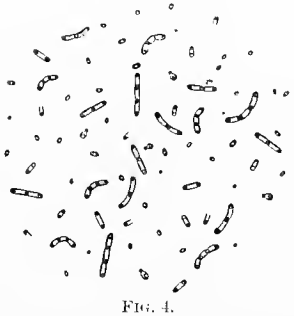


FIG. 4.

Diagramatic. Illustrating the phases of development in a hanging-drop culture of the Germ of Swine Plague, Southern Cattle Plague, and the Corn-Stalk Disease.

PLATE IX.

Fig. I.

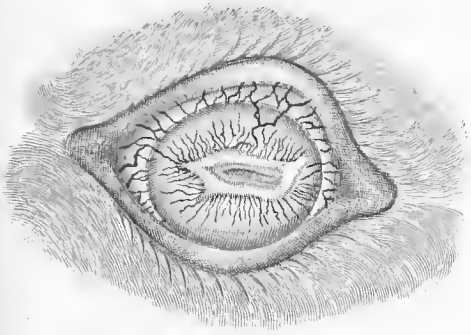


Fig. II.

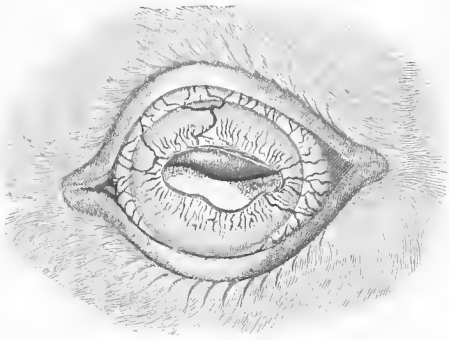


Fig. III.

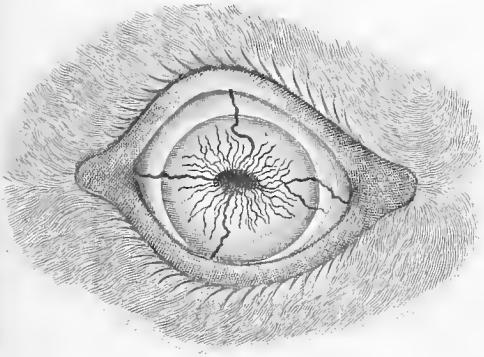
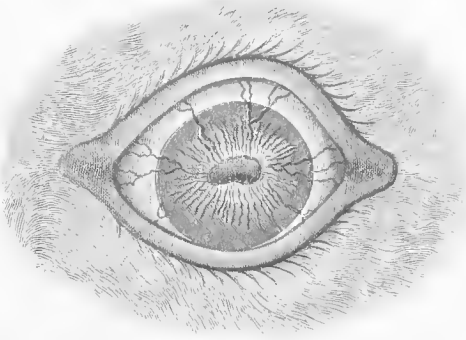


Fig. IV.



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